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Hugo L. Obwegeser

Mandibular Growth Anomalies

**Terminology – Aetiology
Diagnosis – Treatment**

Histology by H. U. Luder

Forewords by P. Tessier and W. R. Proffit

With 102 Figures in 1228 Illustrations,
some in Color



Springer

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*To my family and my profession.
Both have made my life worthwhile.*

Foreword

Was ever a foreword requested for God's words? It is not indecent to write a preface for Hugo's "Growing Mandible"? After training in the Jaw's Old Testament, Hugo is writing the New One that he taught for 40 years long. Indeed, the last decade has been fertile in mechanical gadgets which have made diagnosis and surgery easier, such as, accurate CT 3-D images, and absorbable plating or osteodistractor, which brings a fine touch to treatment timing in children. However, all of this does not encompass the intellectual process, basic principles, tactics and procedures.

In his well-deserved retirement, Hugo has found enough time to write about a small window made through his immense experience. The present book is a unique chance for maxillofacial surgeons to enter into his experience.

This book is not the usual tedious and anonymous compendium of a 50-contributor encyclopaedic squadron, bouncing one over the other without clear conclusions, but rather represents a tiny part of one man's life dedicated to patient care and teaching within a high-level Swiss organization, driven by a clear-minded alpine Austrian who has taught two full generations of enthusiastically motivated oral and maxillofacial surgeons.

No dogmatism at all and far from being a cookbook, chapter after chapter, the descriptions and discussions are strongly supported by the analysis and treatment planning of clinical cases. The reader has the certainty of finding "the case" he is searching for, for his own use.

I could not summarize all the terms of all chapters, and how could I have the competence to present a pertinent analysis of Hugo's "Growing Mandible"? When he concentrated mostly on the mandible and maxilla I concentrated mostly on the maxilla, orbits, and cranium. Rather than pass rough judgement on Obwegeser's book, I am pleased to recall some anecdotes from Hugo's behavior, as distinctive as the footprints of the wild game he loves to track so enthusiastically in the Alps and abroad.

Anecdotes

Personal Regrets: September 1967

I made personal acquaintance with Hugo in Rome in the fall of 1967. However, several years earlier, Jacques Dau-

trely had recommended me to pay a visit to Zurich. I could not do this at that time, but I must say that after my first visit to him in 1968 I deeply felt that I had lost years by not being familiar with his various easy ways of handling the mandible.

Tact: December 1967

A "peer review committee" had been invited to Foch Hospital for a debate on the validity of the newly born craniofacial surgery. Demonstrations were provided for one week to 16 sharp judges among the head and neck specialties (see illustration). They confirmed the viability of the newborn. Upon his departure, Hugo left me a heavy parcel to open after take-off. Inside was the golden "Atos" perpetual clock, which for 33 years accurately and silently informed me that it is time to get up and work.

Friendship: May 1968

The country was in chaos, on strike for two weeks. Aircraft did not fly, trains were stationary, and theatres were deserted. Hordes of anencephalic buffoons were preaching the sixth revolution at street barricades and there was no prospect of it ending. I telephoned Hugo, asking if I might come and see him working with Jean-Paul Delbet, my charming assistant. Affirmative! I left an emergency team running my service. A pilot friend poured the last gallons of gasoline into his Piper Cub tank and flew to Zürich. For a whole week we lived a surgical festival. Hugo had boosted the weekly schedule of his Department in an intensive, attractive fashion. When we returned to Paris, the burlesque "revolution" was out of breath but we were both rejuvenated by Hugo's fresh breeze.

Personal Pride: September 1968

Hugo was spending three weeks in Paris when I operated alternately in two operating theatres and took many bone grafts for orbital fractures and upper jaw deformities. Hugo looked at some of these harvested bones; then he escaped from the operating theatre for a while and returned. At a break I met him smoking a cigarette in the locker room.

Hugo: Do you know, Paul, how long it takes you to take an iliac graft?

Paul: No Hugo; I don't care. I take time with my perfect tools and do not have a Swiss cuckoo singing every 15 minutes.

Hugo: Paul, it takes you exactly the time you need to smoke a cigarette.

Paul: ???! (Cuban cigar smoker).

Hugo: Yes! 10 minutes.

Paul: As much!

Lucidity: June 1969

A workshop in Foch Hospital. After 6 days of surgical demonstrations and follow-up clinics, Hugo said "Paul, what you have shown to 50 of us 2 years now is quite worthwhile. Without more delay you must write your book about the new specialty that you have build up, otherwise you will soon be cheated". PERSPICACITY.

Fair Play: Spring 1970

After a meeting in Paris and a splendid dinner, five friends were strolling down Blvd. Saint-Germain: Karl Eric Hogeman, Bengt Johanson, Jack Mustardé and Hugo Obwegeser: four attackers.

"Paul! You ought to publish your mid-facial advancement-technique".

"Paul! You must publish your intracranial approach to hypertelorbitism and orbito-cranial trauma".

"Paul! You must publish your procedures for rare facial clefts and Franceschetti deformities soon".

"Paul! Please publish your original works in the orbital region. – OH LA LA! Why so much writing?

Unanimously: Paul! Please do realize that we have now acquired our own experience with what you have taught for 3 years and we would like to present our results. But WE cannot write a single page until YOU have published your basic procedures.

In 50 years of medical practice I have never met such a quartet of surgeons singing so well in tune. Therefore, I published in 1970 and 1971 and they did so in 1971 and 1972. That was a group of famous men there on the street. Hombre! All these four aces were true gentlemen – an endangered species, nowadays!

Premonition: 1971 in New York City

There was a discussion about the timing of the reconstruction of the skeletal defects in oto-mandibular dysplasia (H.F.M.). Jack Converse pleaded for the correction of the skeletal anomalies in childhood, Hugo Obwegeser argued for delay until adolescence and he was right. Ironically, 20 years later the bone distraction method has made an average mixture of both.

In recording his experience with the growing mandible, Sir Hugo has accomplished his duty, which I myself have not fulfilled for the upper facial segment. Let us ask him to write another book for more mandibles and the upper jaw.

Paul Tessier



Illustration: Dr. Paul Tessier with his special foreign guests at his a "peer review committee" meeting at Foch Hospital in December 1967. From right to left: Paul Tessier, Ms. X, Jack Mustarde, Karl Schuchardt, Blair Rodgers (representing J. Converse), Roger Mouly, Ms. X, Hugo Obwegeser

Foreword

The diagnosis and treatment of growth anomalies of the mandible improved markedly in the late twentieth century, and the work of Hugo Obwegeser played a large part in that progress. This book is a welcome addition to the literature on the subject, especially because it brings Obwegeser's contributions into a single well-documented source.

Because the author is a renowned surgeon, it is not surprising that the focus of the book is strongly on surgical correction. Professor Obwegeser presents an interesting series of principles that encapsulate the author's point of view and summarize a wealth of clinical wisdom. In addition to the presentation of surgical techniques and case illustrations, the book also provides a summary of etiologic factors in mandibular deformity and Obwegeser's classification of these problems. His differentiation of major types of excessive mandibular growth has had a considerable impact on thinking about problems of this type and is very well presented here.

Mandibular growth problems, of course, are of great interest to orthodontists as well as to maxillofacial surgeons. For the most part, the less severe problems are treated by orthodontists, with a combination of growth guidance and compensatory tooth movement, while the more severe problems require surgical intervention. Almost always, integrated surgical and orthodontic treatment is needed for the more severe cases. This book focuses, appropriately, on the more severe problems that Prof. Obwegeser has treated throughout the course of his long and distinguished career and on the surgical management of these problems, with the role of the orthodontist in the background, not so much ignored as taken for granted.

The primary indication for mandibular surgery in children is a progressive deformity, one that becomes steadily worse as growth continues. The goal of surgery in these children is primarily to establish an environment in which more normal growth can occur, and postsurgical orthodontic growth guidance usually is needed. In this book, Obwegeser reviews growth principles for the mandible, which summarize current views of mandibular growth and its control. The histology of condyles in mandibular growth anomalies is covered

more thoroughly than in any other source of which I am aware. Clinical experience with surgery for severe growth problems at an early age is presented in a series of cases in which the surgical intervention is well documented, but the use of orthodontics for management of subsequent growth is not. The orthodontist and surgeon who work together in these cases will need to be aware of techniques for growth management that are not presented here.

For older patients in whom little postsurgical growth is expected, the goal of surgery is correction of skeletal defects and major distortions of the alveolar process. Presurgical and postsurgical orthodontic tooth movement usually is needed. Prior to surgery, the goal is to establish an appropriate relationship between the teeth and their bony support. Removal of dental compensations for the skeletal deformity allows more complete correction of the skeletal problem. Postsurgically, orthodontics to bring the teeth into their final position has two purposes: it allows greater precision in establishing the dental occlusion than is possible with jaw surgery, and perhaps more importantly, it allows compensation for the small postsurgical changes that usually occur with surgical healing. Because the orthodontic management of these cases is well established and presented elsewhere, the omission of the orthodontic methodology from this book should present little problem.

This book is a valuable reference for orthodontists for several reasons. It provides a unique insight into the thought processes of a creative surgeon, documents the changes that can be produced in a great variety of cases, and illustrates very well the current state of the art in the treatment of mandibular growth anomalies. Orthodontists can enjoy and appreciate the overview of treatment that is presented here, and even more importantly, have a better understanding of the etiology, diagnosis, and clinical course of these problems. The descriptive terminology of jaw anomalies can contribute also to better communication among orthodontists, maxillofacial surgeons, and other dental and medical practitioners involved in the management of these conditions.

W. R. Proffit

Acknowledgements

There are many people who have helped me to write this book. First of all I want to express my gratitude to my wife Luise for her understanding and willingness to let me work on the book for so long, almost 3 years, often for 6–7 h a day, instead of helping her in the garden and to fulfil my promises regarding what we would do together once I had retired. No less I want to thank my very good friend and colleague, Mr. Peter Clarke from Aberdeen, Scotland for his great help in correcting my English. He has done a tremendous job. Without his help I would have had to write the book in German and then have it translated into English, which is rarely really good. I also want to thank my successor and former pupil, Professor H. Sailer, for making the records of my former patients available to me. Without my former secretary, Mrs A. Svoma, this book would never have seen the light of day. She helped me to find as many records and radiographs as possible and she did all the tracing and measuring of all the panoramic view radiographs and many other things. I am also no less thankful to Mrs S. Etter, also a former secretary at my clinic. She did an unbelievable job in typing the text in English, including the often rather large corrections. I am also thankful to the photographers, Mr. Ph. Halioua of the clinic and Mrs L. Brandenberger of the Dental School, and the artist, Mr. M. Haab, for their kind assistance. I am also very grateful to Dr. R. Caduff from the Institute of Clinical Pathology for her efforts to find as many of the histological specimens as possible, enabling Dr. H.U. Luder to write his part on the histology of mandibular growth anomalies. To him also goes my special thanks for finding the time during the very busy work at his institution in order to produce the histological part of the book. I want to thank Dr. Kačl of the Klinik and Poliklinik for Nuclear Medicine for his very valuable help on the chapter on scintigraphy.

Last but not least, I want to thank my friend and colleague, Professor P. Stöckli, Head of the Department of Orthodontics and Paediatric Dentistry at the Zürich Dental School, for his permission to use some of his cases and for his continual support during the preparation of this book. I also want to thank specially his Assoc. Prof. U. Teuscher for his assistance and agreement with my text regarding the principles and terminology and orthodontic considerations.

A very special expression of my thanks goes to my former co-worker, Prof. P. Egyedi from Utrecht, Holland, and to Dr. B. Terry from Chapel Hill, North Carolina, a friend of mine since he trained with me for some months in 1967, for his kindness in reading the whole book and giving me his opinions and suggestions. To my good friend, Dr. Frank Pavel from San Diego, goes a special thank you as he sent my chapter on “Descriptive Terminology for Jaw Anomalies” to Dr. E. Tolman and Dr. M.A. Shampo, Ph.D., at the Mayo Clinic for their editorial help. They did an excellent job and my most grateful thanks and appreciation goes to both of them. Furthermore I want to thank all those colleagues who have referred so many interesting cases to me and for the enthusiastic cooperation in preparing the cases for surgery. I also want to express my thanks and appreciation to Mrs. H. Eschle, in charge of the library at the Zürich Dental School, for her help and efficiency in finding the very old literature I needed. There are many more I have to thank, too many to name them all personally. To all of those who have so willingly given me their help and assistance I want to include in my declaration of my gratitude and appreciation.

Finally, I wish to thank the publisher for accepting my manuscript with so many illustrations and for the excellent appearance of the printed book.

Hugo L. Obwegeser

Preface

The actual spur to write a book on the subject of mandibular growth anomalies came after I lectured on abnormal mandibular growth regulation to the Californian Angle Society in the summer of 1995 in San Diego. The then President of that Society, Dr. Richard B. Laughlin, convinced me of the necessity to have a try. And so did my very good colleague and friend, Prof. Paul Stöckli, chief of the Department of Orthodontics and Paedodontics at the Dental School of the University of Zürich. With him, I have discussed and treated many mutual cases. From these discussions I have learned a great deal.

Most cases with mandibulo-maxillary discrepancies are symmetrical. They generally present no great problem to an experienced surgeon, although even these symmetrical ones are not always all that easy to diagnose properly and to make a generally acceptable treatment plan. The treatment plan may differ quite considerably for a similar case, from one surgeon to another. The making of a proper treatment plan presumes a proper diagnosis “*First diagnose, then treat*” (H.G. Gillies 1920) (Principle Nr. 2).

Much more interesting than the symmetrical mandibular anomalies are the asymmetrical ones. In most of them an aetiological factor could be found. It was either congenital or mostly due to the influence of an adverse factor on the condyle, or a larger part of the mandible, during the growth period, producing a unilateral growth deficiency. But there was also a group for which no aetiology was known for their asymmetric surplus abnormality. These cases had fascinated me since my “professional childhood”. Through a lifelong experience with these cases, I found an explanation for their aetiology. I published this together with our pathologist (H. Obwegeser and M. Makek 1986a,b). The reaction of interest to that publication came mainly from the orthodontists.

Since I have been dealing with jaw anomalies I have heard and read many different terms for the same type of jaw anomaly. The fact that experienced surgeons and orthodontists did, and still do, use different terminology for the same type of anomaly caused me to seek a terminology which describes the anatomical substratum of the anomaly. Such a terminology should be acceptable to all specialists involved in the subject of jaw

anomalies. I already suggested such a terminology in 1986 and 1993 and will include it in a chapter of this book.

After I had decided to go deeper into the subject of these mandibular asymmetries, I found it necessary to also include our clinical knowledge and experience of the facts and factors which influence mandibular growth. The cases in Figs. 11–29 have taught me a great deal on the influence of the condyles on the growth of the mandible. That is the reason for presenting them.

As it is very important to know what should be done, and when, it is definitely worthwhile to go deeper into the subject of mandibular asymmetries from the point of view of their aetiology and the diagnosis as well as the treatment. The principal aim of this book is to attempt to elucidate how those mandibular asymmetries develop for which we do not know an aetiological cause, what kind of variations in their appearance can be observed, and what and when something should be done to them. It is my particular intention to demonstrate these variations on a great number of cases and yet I want to avoid using more than one case to demonstrate a specific problem.

I am still often asked how I perform this or that operation and how I avoid one or the other complication, for that reason I will end the book with a chapter on principal standard operation techniques, and instruments. In that chapter I will reflect the historical background of these procedures, who, when and how they developed. I will describe how I perform these standard procedures and how modern instruments help to make the work easier for the surgeon and how with them he can avoid the formerly rather often experienced complications.

The various facets of the subject mentioned led to the title of the book “Mandibular Growth Anomalies. Terminology – Aetiology – Diagnosis – Treatment”. It is quite a large subject to deal with, but I did so with pleasure and enthusiasm because it is such a fascinating subject.

As a trainee as well as a teacher I learned how important principles can be in dealing with facial anomalies. Whenever possible I tried to formulate important things in diagnosis as well as in treatment planning and execution in the way of clear principles. The most im-

portant ones will be put together in a separate chapter. They will also be mentioned repeatedly within the description of the cases.

The cases I am going to use are samples out of the clinical material collected between 1955 and 1987, when I was in charge of the Clinic of Maxillofacial Surgery at the University Hospital of Zürich and of the Department of Oral Diagnosis and Oral Surgery of the Dental School of the University of Zürich. For some of the older cases presented, panoramic radiographs and scintigraphic examinations and CT-scans did not exist. Many of the interesting and rather instructive cases could not be used as there were only some slides but no remaining records and radiographs.

It is not intended that all the published literature on the subject be dealt with. I am sure the reader rather expects me to tell him of my own experience and opinion on the subject. For that reason it is mainly my own pertinent publications that are cited. However, in dealing with the history of the principal surgical procedures I do intend to cite the pertinent literature in every detail. I will purposely also occasionally quote an older publication on a special subject when I am of the opinion that its statement is still valid today, in spite of other "modern trend opinions".

As the great majority of mandibular growth anomalies start in childhood, this subject is primarily of interest to the paediatrician and in particular to the orthodontist, and also to various surgical specialists such as maxillofacial, plastic and ENT surgeons. Other medical specialities may also be involved from the diagnostic point of view such as radiology, nuclear medicine and even endocrinology.

A special remark on the reproduction of patient's photographs: In demonstrating and discussing the various types of growth anomalies of the mandible and their treatment it is unavoidable, for teaching purposes, to show not only radiographs of the patients, but photographs as well. – By law, everybody has the right that his photographs are not used for publication without his express permission. Otherwise the photograph of the person's face has to be covered in such a way that it is not possible to recognize him/her. As such regulations make it impossible in medical teaching to pass on knowledge in respect of facial anomalies to others, we have to use patients' photographs, which have not been altered. For that reason, all our patients, private as well as state-financed ones, signed on their registration form the written statement that they permit the use of parts of their records for teaching purposes, as long as their name is not revealed.

The purpose of this book should not be only to present information on the subject to all the various medical specialities which are involved either diagnostically or in the treatment. But it should also stimulate everyone to fully document prospectively as many cases as possible to enhance further knowledge on the subject.

I will start the book with a chapter of gratitude to my most important teachers. Without their influence I would not have gained the background and knowledge to write this book.

Hugo L. Obwegeser

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The Basics

How I Became a Cranio-maxillofacial Surgeon

My Debt of Gratitude to My Teachers

If we had to learn everything by ourselves autodidactically just by reading and watching, a school would never develop. An active teaching school is the most rapid and effective means of transference of skills and knowledge and instillation of motivation to further progress, both for the individual and the profession generally. There may be the occasional genius. But the majority of people need teaching in order to assimilate the basic knowledge and skills for their future profession.

When I had completed my medical studies, I started as unpaid visiting doctor number 5 in the small hospital of my rather small home town. The director of the hospital in 1945 was the surgeon, not the administrator. I could watch operations in almost all surgical fields including treatment of fractures of the extremities, then achieved by extension repositioning, followed by plaster of Paris immobilization. I did not know in which direction my medical training should go.

Hermann von Chiari, M.D. (1897–1969) (Fig. A), My Teacher in Pathology and Microbiology

When I discussed my possible future with an uncle of mine, an orthopaedic surgeon who had trained in Vienna for more than 10 years, he said: “whatever you want to become, a general medical practitioner in a small town or a specialist in any field of medicine, you will always need a fundamental knowledge of pathology and pathophysiology”. I wanted to follow my uncle’s advice and decided to go to the Institute of Pathology in Vienna. This was in October 1945. Austria was divided into four occupied districts. I lived in the most western section, occupied by the French. To travel to Vienna, I had to pass the American and the Russian occupied parts of Austria. Vienna itself was also divided into four sections. It took me 3 full days to travel to Vienna by train. Lodging was offered to me by the grandmother of a little boy, a refugee from Vienna, whom I had taken care of while he was receiving treatment in our small hospital for a metastatic osteomyelitis of a femur without antibiotics which were not available in Austria in those days. Through that connection I was certain to have a bed on arrival in Vienna.

The day after my arrival I went to the world famous Rockitansky Institute of Pathology of the University of Vienna. It is that institute where Landsteiner and Wiener had identified the four blood groups. The old building was very crowded. There was not enough room for all the students who wanted to listen to the lectures of Professor Chiari. The autopsy rooms were crowded with students and Austrian and foreign doctors who had returned from the war.

My intention was to train in pathology and microbiology for about 2 years. When I asked Prof. Chiari whether I could train with him he said yes, but in an unpaid capacity only, as there were too many applicants. I became applicant number 10 in an unpaid position. I stood around the dissecting tables and watched and listened. I had no chance to do anything myself for more than a month. Then Prof. Chiari and his coworkers would occasionally let me assist or even do a part of the dissection. Most of the cadavers were elderly patients who had died of pneumonia or a cardiac problem or of cancer. A lot had suffered from malnutrition. Every day we received a cup of Russian peas. That was our daily

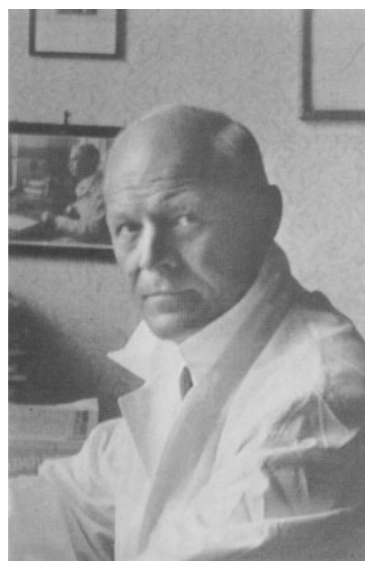


Fig. A. Professor Hermann v. Chiari

food ration. Whatever was available on the ration cards was not enough to survive on. Additionally, one had to have money to buy something on the black market. As I had no salary I had to find other ways to earn money. The black market was the great chance. It did not take me long to find out that I was absolutely untalented for earning that type of income. So, I decided to give pre-examination courses to students as a help in preparing them for the final examination in microbiology and general pathology. These courses were, and still are, quite common in Vienna. By doing that I earned the necessary money to pay for the bit of rationed food and for some additional food from farmers.

After working at that institute for a year as a visiting doctor I was promoted to a paid position. Pathology and pathophysiology fascinated me. Prof. Chiari liked my enthusiasm. One morning there was a cadaver of a 66-year-old patient who had died from Henoch-Schönlein purpura. Prof. Chiari invited me to find the aetiology of that disease as it was still unknown. Three possibilities had been discussed. I had to take specimens from every organ and in particular from the intestine with its severe bleedings from which the patient had died. The technicians prepared all the histological sections with the different types of staining which I wanted. I sat with them in my study, studying them under the microscope every day. I couldn't find anything suggestive of a possible cause for the severe bleeding in all organs and tissues. After 3 months Prof. Chiari offered me half an hour to discuss the problems of the case with him. He also searched through many slices. Then he said: "I do not find anything myself either, but the patient died of that disease. Go back into your study and find the aetiology". I looked through these slices every day again and after 3 more months I went to Prof. Chiari once more and told him that I still could not find anything. He himself put a number of slices under the microscope and found nothing either. Again he said: "The patient has died of that disease and you will go back into your study and find the cause". Every day I searched for something which I did not know. After some more months I found, in many slices of all different organs, skin, intestines, brain, kidneys, heart, musculature, etc. all the classical stages of allergy of the small arteries. Prof. Chiari confirmed my findings and agreed with my diagnosis. I was then very proud of having discovered the aetiology of this disease (Obwegeser 1953).

Pathology and pathophysiology had become like a crime story without an end to me. I wanted to stay on. But circumstances caused me to find another field of interest in medicine. Almost the whole staff of the Institute of Pathology in Vienna became infected by some type of tuberculosis, some of the lungs, as the food available in those days was very insufficient and others acquired skin tuberculosis of their hands as we had no rubber gloves for use when dissecting the cadavers. After

2 interesting years of learning under Professor Chiari I thought I ought to leave before I myself acquired such an infection. In those days (1947) there were still no antibiotics available against tuberculosis in Austria. - I had experienced the efficacy of penicillin when I worked in the section of microbiology of that institute. It was in 1946 when an American team demonstrated intubation anaesthesia, for the first time in Vienna. The team had also brought penicillin. They injected a dose of 50,000 units to a patient with chronic gonorrhoea from whom the section of microbiology of our institute had cultivated the bacterium *Neisseria gonorrhoeae* as proof of the disease. With one dose of 50,000 units of penicillin the patient was cured of his disease. Unforgettable!

I wanted to train in internal medicine. I thought my background in general pathology would give me a good basis for that speciality, which I considered as the queen of all medicine. In spite of my former training in general surgery for almost 1 year and 2 years in pathology and the support of Prof. Chiari I received only a negative reply to my application for a paid training position in one of the two clinics of internal medicine at the University of Vienna. My next choice was gynaecology and obstetrics. I received the same negative reply. Everywhere there was a long list of applicants.

Richard Trauner, M.D., D.M.D (1900–1980) (Fig. B), My Teacher in Maxillofacial Surgery

At the Institute of Pathology I was responsible for the photomicrography. In 1946/1947 the equipment was still old-fashioned. Photomicrographs were taken in the



Fig. B. Professor Richard Trauner

same way as was done before the war. We also made them for other specialties. Richard Trauner, a coworker and nephew of Hans Pichler, the famous pioneer in our speciality, who influenced the development of maxillofacial surgery with his Viennese school not only in Austria and Italy, but also in all the east European and the Balkan countries, wanted to publish his work on fibrous diseases of the jaws. For that he needed photomicrographs. That was how we met. He had just been appointed chief of the Dental School of the University of Graz with its maxillofacial surgery department. He offered me a paid position for training in maxillofacial surgery if I would agree to come with him to Graz University. Besides, I could do my dental training for the Austrian dental qualification. In those days dentistry in Austria was a subspeciality of general medicine which required two additional full years of dental training followed by an examination. I accepted Trauner's proposal, as dentistry offered me the fastest way to earn my own living. I had no intention of staying on in maxillofacial surgery. I wanted to get married. This is why I needed a safe income. As I found more than average training important to establish myself in the profession, I did additional training in all fields of dentistry as well as working in maxillofacial surgery.

R. Trauner opened our eyes regarding the scope of our speciality and stimulated us to see problems and find solutions for them. Through his enthusiasm for the various fields of maxillofacial surgery I also became more interested in it and continued with my training. It had never been my intention to become a university teacher. Finally I was on the road. Trauner was far-sighted. He sent us to other places to watch and learn. He himself had great experience in jaw pathology. He was also well known for his cleft lip and palate work.

At the department of maxillofacial surgery of the University Hospital at Graz, Austria in those days, the main subjects were primary clefts, cancer cases and any number of patients with facial trauma and there were also still some cases with severe facial war injuries. Altogether I trained 6 years with R. Trauner. He was not only a teacher to us trainees but also like a father.

Sir Harold D. Gillies, M.D. (1882–1960) (Fig. C), My Teacher in Plastic and Reconstructive Surgery

To improve the soft tissue reconstructive work on our war and cancer cases, Trauner sent me to Sir Harold Gillies. The British Council enabled me, with a scholarship, to learn from the founder of modern plastic and reconstructive surgery.

In October 1951 I went to Basingstoke, south-west of London, where Sir Harold worked at the Plastic and Jaw Unit at Rooksdowne House, an old mental hospital. Pri-

marily he was a specialist in ENT who developed an interest in facial reconstructive work. He worked mainly on the reconstruction of war cases, together with Norman Rowe who was in charge of the oral surgery section. My knowledge of the English language was what I had learned at school and never practiced for 13 years. Sir Harold was a New Zealander with a heavy Scottish accent. I could not understand what he said. There was also a Norwegian oral surgeon, Haymann Krømer, as a trainee who had already been with Sir Harold before for a longer period. He spoke perfect English. I could understand him a lot better. After 3 days he asked me where I came from. When I told him that Richard Trauner from Graz in Austria was my chief he asked whether he was that Trauner who had published three volumes on oral and maxillofacial surgery together with Hans Pichler. After my positive reply his face became very friendly and from then on he spoke to me in perfect German. He had done his dentistry in Leipzig. Often we played table tennis together and were very good friends until he died, much too early.

At Rooksdowne House I also met Ralph Millard and Ivo Pitanguy and many other foreigners, who later became rather famous plastic surgeons in their home countries where they built up their own schools, passing on to their pupils what they had learned from Sir Harold.

Sir Harold was a genius. He was a brilliant teacher, very clear and systematical. While watching him during his operations, he asked me to write down the principles which came into his mind while doing his work (H.G. Gillies 1952; H.G. Gillies and R. Millard 1957). From him I learned how to plan the reconstruction of a facial defect and how to handle soft tissues. He liked to teach in



Fig. C. Sir Harold Gillies

principles, many of them are still of general value in any field of surgery. *"Before planning the reconstruction of a defect, replace into normal position what is normal and retain it there. That allows you to see the actual defect"*. He hated the use of toothed forceps for holding a skin flap. *"Every grip with it will kill cells. Use fine hooks only"*. He taught us how to undermine the skin to create a much larger flap. When I came home I used that undermining technique in producing a new procedure for vestibuloplasty in preprosthetic surgery. I called it "the submucous vestibuloplasty procedure". From Sir Harold I learned so many principles and techniques that without the training with him I could not have treated so many difficult cases as successfully as I did.

It was fascinating not only to learn from him professionally but also privately. As I could not afford to travel home to Austria for Christmas he very kindly invited me to his family Christmas party. I was very grateful that I did not need to be alone on Christmas Eve. Occasionally he took me to his home and we worked together on his very small private golf course. For professional reasons I would have loved to have stayed on for a longer period, particularly as he had invited me to join together with him and Ralph Millard in writing the book they had planned on "Principles and Art of Plastic Surgery". After 5 months I had to leave for home as my child number three was already 3 months old and had not yet seen her father. I met Sir Harold later again several times. I will forever remain grateful to him.

Eduard Schmid, M.D., D.M.D. (1912–1992) (Fig. D), My Teacher in Technical Details

Eduard Schmid had trained with Wassmund for 2 years and started after the war from nothing to build up a department of facial reconstruction at the Marien-Hospital in Stuttgart. Trauner had sent me to him twice for a month. According to the results in reconstruction of facial injuries from the war which he had shown at annual meetings of the German Association of Maxillofacial Surgery, he seemed to be the best man in this field in the German-speaking area. He could sit beside a patient for an hour, still seeking the best way to produce what he wanted to achieve. From him I learned that with delay operations the blood supply could be trained to go where wanted.

Unforgettable, how he used the whole covering of the pharyngeal wall to reconstruct a total defect of the soft palate. He placed thin slices of perforated cartilage underneath that pharyngeal mucosa. Three months later he raised the mucosa including the cartilage and an adherent soft tissue layer and put mucosal grafts, taken from the cheeks underneath, as a second lining for that flap which later had to become the soft palate. When a few weeks later he fixed the pharyngeal flap to the pos-

terior rim of the palatal defect, that flap could not shrink, neither in length nor in width. With that type of pharyngoplasty the patient could then speak again. This technique of training the blood supply I used when a trainee of mine had managed to damage the whole palatal mucosa in a cleft case so badly that it necrotised completely. He had tried hard to mobilize that cleft maxilla with two Rowe's disimpaction forceps. The cleft was open again the full length of the hard palate. I covered the raw palatal bones with vaseline gauze, kept in place by an acrylic plate.

After some weeks spontaneous secondary epithelialization covered the bony parts. It was a mucosa without any submucous tissue, of no use to produce flaps to clothe the open palatal cleft. With the technique of Eduard Schmid I managed, step by step, to get a blood supply to that mucosa from the soft palate. After a few delay operations I was able to raise the secondarily epithelialized mucosa as flaps on both sides for closing the cleft defect.

Eduard Schmid was not only a master in soft tissue matters but also a pioneer in bone work. In maxillary defects, he first reconstructed the missing soft tissue, mostly with local skin flaps and then implanted iliac crest bone via the oral route to build up the maxilla so that it could support a denture again. This was before antibiotics were available to him.

It seems hardly understandable that nowadays an extraoral approach is still frequently used for reconstruction of defects of the horizontal and ascending rami, producing scars on the face when they are not necessary.

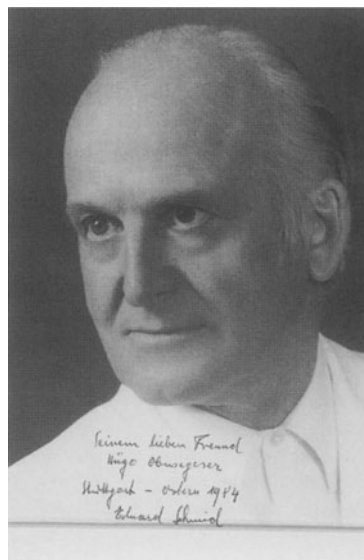


Fig. D. Professor Eduard Schmid

Paul Tessier, M.D. (Fig. E), My Last Teacher and Personal Friend

A good way to learn is by watching others. Those I would not call my teachers. My teachers are persons from whom I learned very important principal techniques. From Paul Tessier I have learned the principal technique for advancing the whole middle third of the face and also how to rotate the eye sockets into the planned position. No other surgeon working on the facial skeleton has opened such new important possibilities for correcting anomalies, posttraumatic or congenital, of the upper half of the facial skeleton as he has done. As mentioned later, I worked on the Le Fort III advancement myself and I guess, I might have finally succeeded, but I am not sure whether in such a clear way as Paul Tessier did. In addition to that, his cranial approach to the orbits via the anterior base of the skull has created the possibility of producing a normal appearance and with that a normal life for many patients. Before these new techniques were developed, people were condemned to live with the disfigurement they had. His new procedures were the highways for further ideas and improvements, some by himself and some by others.

What a wonderful gift it is that nowadays we can make normal faces out of many very disfigured ones. This is due to the fact that we can correct any deformity in the lower half of the facial skeleton as well as in its upper half. For the fact that Paul Tessier taught me his procedures shortly after having developed them, I will always be grateful to him and call him my last teacher. Why a surgeon who has created such procedures which enable him and many others to give prerequisites to normal life to so many patients should not be eligible for a Nobel Prize I do not know. In my and many other surgeons' opinion Paul Tessier's ingenious surgical developments and innovations would well deserve it.

In my opinion it is due to my fundamental basic training in medicine and dentistry and due to my teachers' influence that I was able to produce new ideas and procedures, some of them in contrast to the then existing rules. The influence of my teachers enabled me to become a teacher myself. It enabled me from 1958 until 1987 to establish the so-called Zürich-School of Maxillofacial Surgery.



Fig. E. Monsieur le Docteur Paul Tessier and the author, photograph taken at the occasion of the founding of the European Association of Maxillofacial Surgery in 1970 at Zürich

Introduction

The facial appearance is the result of the size and position of its various bones and how they fit together and the investing soft tissues. The mandible plays a special role in this appearance. It is the only moving part of the facial skeleton and not only of special importance for chewing and speech, but also in the aesthetic appreciation of the face and its expressiveness. Its form in its resting position as well as in its movement is very important in its assessment. The size and position of the maxilla is also very important from the aesthetic point of view as well as function. The growth of the mandible and in particular its growth and developmental anomalies can influence the maxilla in its development and when abnormal can have a very negative influence on chewing and speech function.

It is evident that the zygomata, the nasal framework and the forehead and the covering soft tissues also play their part in the appearance of the face. If they are divergent from the normal, the patient may also feel handicapped. However, they do play a much less important role in form and function than does the mandible, especially in its form and its position relative to the maxilla.

Because of its incomparable variety in abnormal positional and dimensional possibilities, the main interest in treatment has long been focussed on the mandible. Today we are in a position to correct very satisfactorily almost every anomaly of position and shape of the mandible and the other parts of the facial skeleton without facial incisions.

Correction of the position and form of the mandible must always lead to a result in which the teeth are in good occlusion with those of the maxilla. For that reason, almost all cases of anomalies in shape and position of the mandible are a surgical as well as an orthodontic problem in treatment planning.

There is no other bone in the whole human skeleton which can produce so many different typical growth anomalies as the mandible. Abnormal growth of the mandible leads to an abnormal shape and position, thus producing a more or less pronounced facial disfigurement. This may be due to the anomaly of the mandible only or, in addition, also due to a secondary abnormality in size, shape and position of the maxilla. While an abnormality of the mandible can have a strong influence on the development of the maxilla, the reverse does

not apply. Even a very severely deformed maxilla as occasionally still found in secondary cleft deformities, may not have the slightest influence on the final shape and position of the mandible. This interesting fact makes it even more important to know the variety of the mandibular growth anomalies and to understand their pathophysiology.

The fact that no other bone in the whole human skeleton is capable of producing such a great variety of typical growth abnormalities as does the mandible, is partially due to the fact that it consists of two halves which normally develop symmetrically, comparable to the long bones of the two halves of the body. The two halves of the mandible ossify in the midline of the face, in the so-called mandibular symphysis, during the second year of life, in such perfection as if the mandible were one single piece of bone; only because of that may abnormal growth and growth development of one side consequently also influence the other side in its form and position. On the other hand the great varieties of mandibular anomalies definitely depend on the very complex growth mechanism of the mandible. There is no doubt that each half of the mandible has its own independent growth regulation capacity which makes it possible that entirely different halves may develop, even without the influence of any known agent.

As there are several possible causes for the development of an asymmetric mandible, the various aetiological factors are of great interest. However, I do not intend to discuss and demonstrate cases affected by each possible known aetiology.

It is not always easy to make the right diagnosis immediately, although the deformities are so clearly differentiated in their anatomical structure. And yet elongation of the one half of the mandible may be diagnosed as such because the other half is shorter. However, it might be that the reverse is true!

For most mandibular asymmetries an aetiology can be found in the patient's history and in the radiological appearance of the mandible. But also asymmetrical mandible cases have always been observed for which no aetiology was known. Occasionally we saw the case with a mandible in which the affected side of the face was vertically clearly longer than the normal side (Fig. 1a) and we saw rather frequently the type then called late-

rognathia or asymmetric prognathism (Fig. 1b). And even less commonly we saw the patient with a very twisted mandible producing a really very unpleasant facial appearance (Fig. 1c). This overgrowth may start at any time from childhood up until growth has ceased or even afterwards and these mandibular overgrowth asymmetries may be found in any degree of severity. For all other types of mandibular asymmetry an aetiology could usually be found.

For most of the patients, the reason for seeking help was the anomaly of the shape of the mandible or its asymmetry or even the asymmetry of the whole face. In some cases TMJ clicking or pain was the reason why the patient sought help. Some mentioned a possible trauma as the reason for the mandible to grow asymmetrically. If that were the case, the abnormal growth would have to have started soon after that trauma to the face or the chin and not several years later.

Not in every case to be demonstrated will the patient's general condition be mentioned. Only in those patients in whom there was obviously something wrong with the general health situation will I mention it.

Occasionally a hereditary aetiology was considered possible. This could only be true when the anomaly started during normal growth and not after general growth had ceased. The inherited anomalies of the mandible are never unilateral. The genetically fixed anomalies of the mandible are, in my opinion, not caused by misregulation of growth but are due to the genetic pattern they have to follow.

The cases I am going to use in this book were either operated upon by myself or by one of my trainees according to my planning. In most of the cases treated by

corrective osteotomies, intermaxillary fixation and wire osteosynthesis were used as plates and screws for osteosynthesis in maxillofacial surgery were not then available in proper quality. This, however, did not influence the diagnosis nor the treatment planning and the result.

Most of my cases came from abroad seeking diagnosis only or treatment as well. For that reason most of the cases did not receive proper presurgical orthodontics or even none at all. I will understand that many of those who read this book may want better final occlusal results and better arranged dental arches. I apologize for the fact that for teaching purposes I had to include many cases which I had to operate upon in spite of a lack of or poor, pre- and postsurgical orthodontics.

I am certain all readers will agree that for the patient with a mandibular or facial asymmetry the most important wish is to have a normal outer appearance independent of whether he can find and afford a good orthodontist. "*Patients with a facial anomaly primarily wish a normal appearance*" (H.G.G. Gillies 1952) (Principle Nr. 1).

The documentation of the patients was done within the teaching programme of quite a large number of trainees. It is therefore understandable that often it is not complete. In many cases of insufficient records, I telephoned the patient in order to obtain more precise details of the patient's age when the anomaly was first noticed and of a possible cause.

The subject of mandibular growth is rather complex. This is also the case with the variety of its manifestations. For these reasons I will use quite a number of cases to demonstrate what is the clinician's experience with the subject of mandibular growth and even a greater

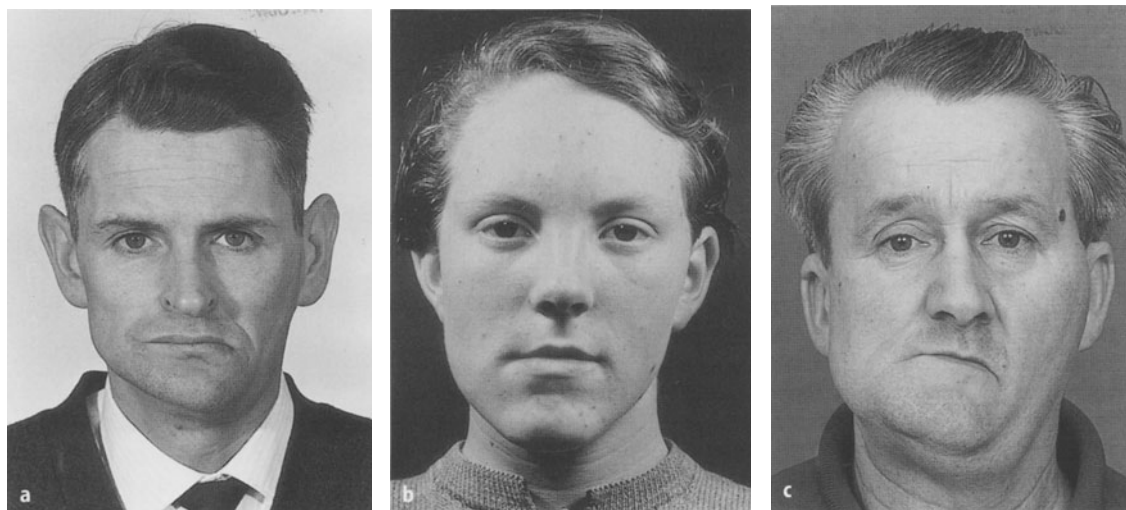


Fig. 1a–c. The three typical forms of mandibular growth misregulation due to unilateral condylar hyperactivity. **a** Appearance of hemimandibular hyperplasia (H.H.). **b** Appearance of hemimandibular elongation (H.E.). **c** Appearance of hybrid form of H.H. and H.E.

number of cases to demonstrate the variety of manifestations of growth anomalies.

In the diagnosis and treatment planning, as well as in the execution of the necessary therapy I have learned from my teachers Richard Trauner, Eduard Schmid and in particular from Sir Harold Gillies, that the ability to adhere to proven principles is a great help. For that reason I have always tried to formulate my knowledge also in principles applicable to our field of work. I have often used them in postgraduate courses, in my daily teaching of trainees and visitors and in lectures. These and some of my teacher's principles will be incorporated in the diagnosis, planning, description of the cases and in the treatment.

The title of the book already defines its purpose. It has four goals. One is a clear terminology. We must have such a clear terminology for the various form anomalies of the jaws. The terminology used should enable all specialties involved in the diagnosis and treatment to speak to each other in the same language. Secondly we want to know the aetiology of these growth anomalies. That means we want to understand their pathophysiology. I do hope that this subject will become clear for the reader by the demonstration of a great many possible variations. Thirdly, proper diagnosis is mandatory for treatment planning of any case of pathology. The large number of cases included should help to improve the reader's ability in diagnosing these mandibular growth anomalies. The fourth goal is the treatment of these cases. I will only give general treatment guidelines for

both the orthodontic as well as the surgical treatment. In every case which had been operated upon I will indicate what the treatment plan was and what was actually done.

I do not intend to discuss in every case why I planned the surgical correction of the anomaly in the way I executed it. I have learned that almost every surgeon has a better plan than his colleagues and also does the surgery better! Nowadays I might correct some of the cases differently than when I actually treated them. We should not forget that nowadays we often perform a bimaxillary repositioning. Repositioning the maxilla as we do it now has been used for about 40 years and it was in 1966 that I popularized that procedure for the first time during a postgraduate course at the Walter Reed Hospital.

Only in some of the cases will I report all of the details for didactic reasons. In many cases only those findings will be shown which are essential to make the diagnosis.

As the patient generally comes for improvement of his outward appearance it should be the experienced surgeon who suggests what kind of profile line and facial shape he wants to achieve. For that, he will have to move parts of the mandible and/or the maxilla into a position necessary for the desired result. The orthodontist will have to be asked for his help in arranging the dental arches in such a way that the surgeon can achieve a good outward appearance without being compromised in doing so because of the position of the teeth.

Aetiology of Jaw Anomalies

The shape and position of the jaws is mainly genetically predetermined, although one has to accept a certain latitude in variations from what we may call a “normal” shape and position of the jaws. In spite of this, there are a large number of positions and shapes of the jaws which cannot be called normal. The causes of these anomalies can be classified into a very few groups. Each jaw anomaly can always be diagnosed as belonging to one of these groups:

- Genetic pattern
- Embryonic disturbance of growth
- Postnatal damage before or after growth has ceased
- Abnormal growth regulation after birth
- Other aetiologies

2.1

How to Diagnose Jaw Anomalies

The diagnosis of an anomaly in shape and position of the jaws cannot be done in an abstract way. It is only possible in relation to the whole form of the facial skeleton, from the front view as well as from the profile view. The form of the upper and lower jaws and their relationship to each other determine the profile in their relation to the base of the skull and the forehead. The three classical profile types, the Roman-Greek profile, also called the straight retroface (retroprofile), has to be accepted just as well as the so-called straight anteface (anteprofile) or also the middle value face which I call the vertical profile. In these three ideal profile types, the upper and lower jaws in their size and shape are in an ideal relationship to each other, however, in a different antero-posterior relationship to the prominence of the forehead. Which one is the ideal profile? It is mainly a problem unique to the patient, and for the diagnostician and the patient's acquaintances to answer that question. Some may favour the straight anteprofile better than the straight retroprofile or the vertical profile. The form of the face and the profile should always be in harmony with the general skeletal form of that person.

During my long clinical career, I have seen repeatedly the same or very similar deviations from the normal shapes and positions of the maxilla and the mandible in the symmetrical as well as in the asymmetrical forms. For the symmetrical aberrations in size, form and posi-

tion of the jaws, there was always the explanation of genetic predisposition independent of whether it was an ante- or retroposition or of a maxilla or mandible of normal or too large or small a size. With this argument, one could also explain the form of the body of the mandible, its ascending ramus and its gonial angle. The size and shape of the prominence of the chin seems to have the same aetiology and in addition they can be influenced by the anomaly of shape of the symmetrical form aberrations of the mandible.

For the asymmetrical forms, this explanation is not valid. Almost all of them have a clearly recognizable cause as long as they develop postnatally. The aetiology of those developing during embryonic growth is only partially known. However, there are embryogenetic as well as postnatally developing forms for which the aetiology is not known. The clinician has finally to deal with all groups. He is naturally very interested to know the cause in all types in order to be able either to prevent their development prophylactically or to “shift the switch” to allow early normal growth and development in the shape of the jaws to occur. These asymmetrical types are the ones which are so interesting to us because of their great variety.

2.2

Descriptive Terminology for Jaw Anomalies

2.2.1

Problems with the Current Terminology

The existing terminology for positional and shape anomalies of the jaws has never really satisfied me. Just a few simple examples prove the unacceptability of the commonly used terms. Early in my career, one learned to speak about prognathism or mandibular prognathia without considering the position and size of the maxilla. This term includes the possibility of a real forward position of the mandible as well as a pseudo-forward position in the case with retroposition of the maxilla. Independent of the fact whether in such a case the maxilla is moved forwards surgically or the mandible backwards, or both towards each other, the resulting occlusion will always be the same but the resulting appearance of the face can differ very markedly. Since, in most

such cases, the desired result with regard to the appearance and occlusion can be achieved only with close cooperation between orthodontist and surgeon, both specialities should use the same terminology. For both clinicians the anomaly has the same skeletal and dental abnormalities. However, no encompassing terminology common to both specialities exists. Orthodontists use the Angle classification, which describes the occlusal situation only. If I hear surgeons talking about correcting a class III case, I feel pain in my professional soul. The surgeon should use a terminology which describes the actual skeletal anomaly. And yet surgeons often use different terms for the same anomaly, not only in different languages but often even within the same language group.

For some years now, American surgeons have come closer to a more descriptive terminology by talking about mandibular or maxillary deficiency and excess. However, that terminology is not so precise that orthodontists as well as surgeons can immediately picture the case without actually seeing it.

Since, in all specialities in medicine, the imperative is “*first diagnose then treat*”, as formulated by Sir Harold Gillies (1952) (H. Gillies and R. Millard 1957), this principle is also true for planning the treatment of jaw anomalies. Until now, there has been no terminology which clearly describes the morphology of dentofacial anomalies. In 1969, I published a paper (H. Obwegeser 1969) on the correction of what I termed micromaxillism and retromaxillism. These neologisms are a clear description of either the anomaly of the maxilla or its aberrant position, and they are now frequently used. Again in 1986 we attempted to put emphasis on the terminology and explained it schematically (H. Obwegeser and M. Marentette 1986).

To me it seems obligatory to establish and use a terminology which describes the anomaly diagnostically, thus enabling both the orthodontist as well as the surgeon to communicate unequivocally (H. Obwegeser 1993, 1995). This proposed terminology usually indicates where the correction has to be performed.

2.2.2 What Is Normal?

Before one can discuss anomalies one should first know what is normal. This is the great problem, for we do not have a single shape, size and position of the facial skeletal parts which we generally accept as normal. Again, what might be called normal in the Caucasian race may be termed abnormal in another race.

The orthodontists have invested quite some effort in evaluating the various profile types. It was A.M. Schwarz (1951), an orthodontist from Vienna, who classified three profile types. He called them “Gerades Vorge-sicht”, “Gerades Rückgesicht” and “Mittelwertsgesicht”.

For this classification the patient's profile line is brought into relationship to the soft nasion vertical line (SNV) and the pupillary vertical line (PV) to the Frankfort plane. As we are evaluating and classifying the various facial forms in their profile line I prefer to call them accordingly “*Straight anteproof*”, “*Straight retroprofile*” and “*Vertical profile*” (Fig. 2). I find that latter term justified, as in this profile type the soft nasion point and the subnasal point lie on a line almost vertical to the Frankfort plane.

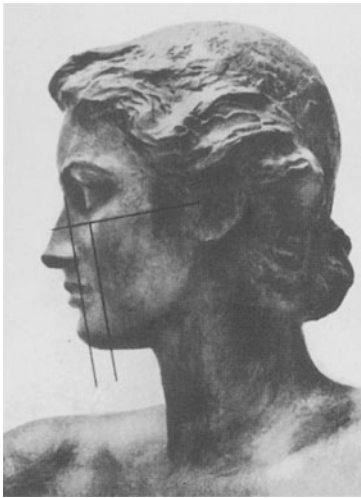
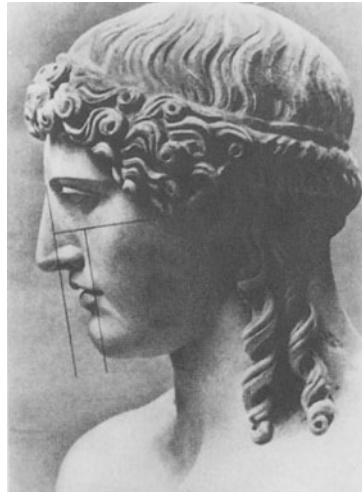
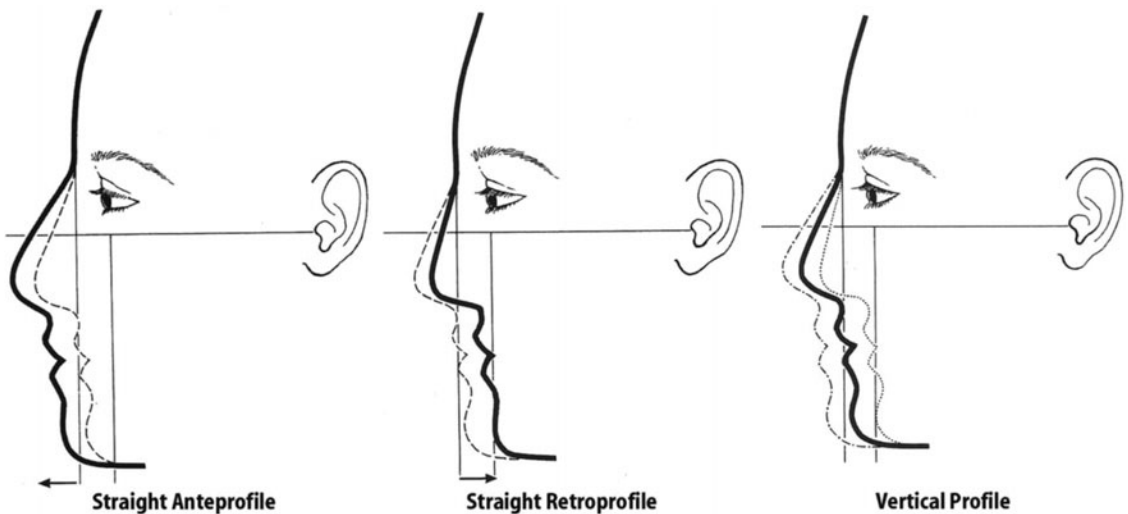
The patient's face has to be evaluated, however, not only in profile but also in its width. From the latter point of view we may use the terms wide, medium or narrow, again in relation to each of the three thirds of the face. The zygoma may be large (hyperplastic), medium-sized or small (hypoplastic).

The anomalies of the frontal bone and of the nose will not be included in this discussion.

Evaluation of the position and form of the jaws cannot be done without relating them to the other parts of the facial skeleton. This is true for the maxilla as well as for the mandible. Since the opposing jaw can also be in a correct or incorrect position, the diagnosis must firstly depend on the particular profile type the patient in question desires, or should have. Into that profile type, the upper and the lower jaw must be positioned properly, independent of the teeth. It is the form and position of the bases of the upper and lower jaw which are mainly responsible for the patient's appearance and not the position of the teeth. For this reason the following principles are the key to the treatment planning of a case: “*The size, form and position of the bases of the facial thirds determine the profile type. Therefore, a real alteration of the facial profile can be achieved only by displacing the bases of the facial thirds*” (Principle No. 6). “*When planning the correction of a maxillo-mandibular anomaly, consider the jaws as if they were edentulous. Then you will be able to bring their bases into the proper relationship to the desired profile and not just the teeth*” (Principle No. 10). The evaluation has to be made from that viewpoint. The diagnosis of the shape, size and position of the jaws is obligatory for defining the profile type which suits that particular patient.

By *normomandibulism*, I mean the body of the mandible is in form and position in that profile type, which best fits that patient. This means, consequently, that *normomandibulism* in the case of a vertical profile face has the mandible in a position which, in the anteface profile, would be a *retromandibulism* and in the retroface profile it would have to be diagnosed as an *antemandibulism*. For the maxilla, the determination of its position has the same prerequisites (Fig. 3).

In order to be able to classify or name the various anomalies of the jaws for our evaluation we must have a clear vision of what the normal should be for that specific patient.

**GALATHEA VON KLIMSCH****KASSELER APOLL****BAMBERGER REITER****Fig. 2.** Drawing of the three classical profile-types by A.M. Schwarz, compared to classical historical figures (from L. Kirchner 1961)

May I suggest that one describes as normal that facial form which is harmonious with the total habitus of that patient. We might call it good-looking or even beautiful. For this reason we do not have any real measurable evaluation. The cephalometric evaluation does not give us the information which profile type is best for that particular patient. We all know people with a classic straight ante- or straight retroprofile or a vertical profile whom we would prefer to see with another profile type and yet the cephalometric assessment does not tell us this. A simple example: a so-called Class III or mandibular prognathism appearance is definitely as unappealing and ill-suited to females as is a Class II or mandibular deficiency in males. In both, the profile will be en-

tirely different whether we move the maxilla forward in the Class III case or the mandible backwards, or even both. And in the Class II case it is the reverse.

So the decision as to which facial skeletal relationship is normal for that patient remains a personal decision for both the surgeon who plans the correction and the patient. It should be discussed and mutually agreed upon.

2.2.3 Need for a Descriptive Terminology

From the surgeon's point of view, in planning the correction of a case with skeletal facial anomalies and also

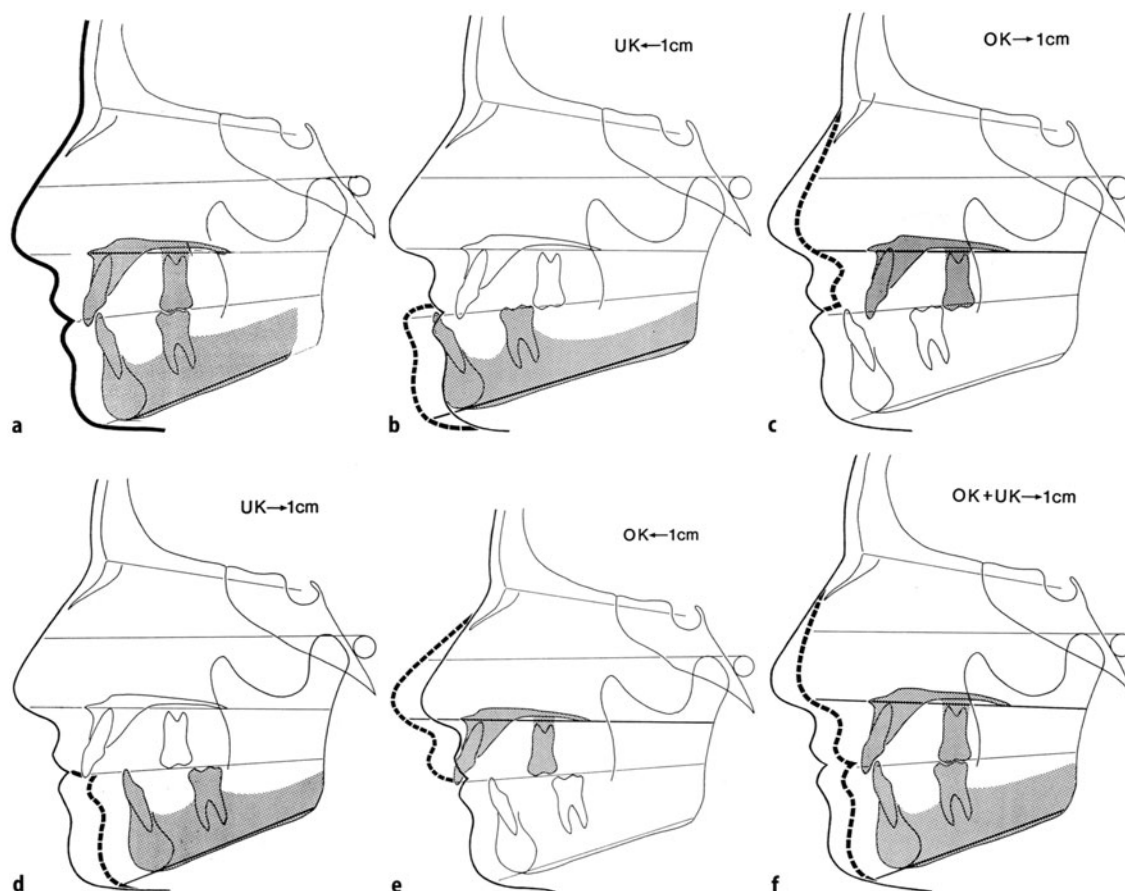


Fig. 3a–f. The diagnosis of the position of the bases of the jaws depends on the patient's profile type (from H. Obwegeser and M. Maren-tette 1986) **a** Normomaxillism and normomandibulism in a case of straight anteproof, **b** when the mandible is moved forward, an antemandibulism (prognathic) appearance will result, **c** when the maxilla is moved backwards the occlusion will be the same as in **b** but the profile of a retromaxillism or antemandibulism results; **d** when the mandible is pushed backwards the profile of a retromandibulism or antemaxillism results; **e** when the maxilla is moved forwards the occlusion will be the same as in **d** but an antemaxillism profile results, to such a degree that it becomes obvious that the alteration had started from a straight anteproof type, **f** when the upper and lower jaws are moved posteriorly by the same amount, the occlusion will be the same as in **a**, but a vertical profile will result out of the straight anteproof

in order to be able to communicate with each other and with other specialists (orthodontists, speech therapists, paediatricians, surgeons, radiologists, nuclear medicine, prosthodontists etc.), we must use clear terms for whatever we want to talk about. This is not only true for the variations and anomalies of every single bone of the facial skeleton but it is also true for the variations in their positions relative to each other. A clear terminology is also necessary for describing the various parts of each individual bone of the facial skeleton. This becomes very obvious when we have to deal with and discuss the mandible.

In the literature, different terms are used for the same parts, in particular those of the mandible. This is only true not for the parts themselves but also for the adjectives

used to describe the form of a part. As already mentioned, the lack of a clear terminology becomes very obvious if we want to communicate regarding positional anomalies of the upper and lower jaws. For these reasons I will try to suggest a clear unambiguous terminology to be used when talking about maxillo-mandibular anomalies.

I do not expect that everybody will agree right away with my terminology and my classification of the anomalies of the jaws. The whole matter may sound strange to one who has never been taught using these expressions. I am of the opinion that we must use a terminology which accords with our diagnosis just as is the custom in general medicine.

Parts of the Facial Skeleton

In order to make things easily understandable I will use the following terms in the discussion of the subject. I will talk about the *mandible* meaning the whole lower jaw and about the *maxilla*, the upper jaw. From my point of view the *chin* has to be evaluated separately, as do the zygomatic bones (*zygoma*) or the nasal framework (*nose*) or the *frontal bone*.

Primarily it seems very important that in the terminology one distinguishes between the base of the jaw, and the alveolus and the occlusion. Therapeutically, there are several different anatomical sections of each jaw which have to be considered. In spite of a normal mandibular base, and on top of it a normally formed and situated alveolus, an occlusal anomaly may exist for solely dental reasons. In this description I will not include any malpositioning of the teeth within an alveolus which is normal in shape, size and position on the base of that jaw. I will only include anomalies of the bony parts of the jaws and will talk about the angulation of the teeth in relation to their pertinent jaw base.

The term “normo-” will be used to describe the maxilla, the mandible, the chin, the zygomas, the nose and the frontal bone which are normal in size, shape and position and in relation to the other facial skeletal parts and normal for that specific patient. With other expressions a specific name will refer to what, in my opinion, is abnormal.

So all the terminology to be suggested has, as a prerequisite, the necessity to first establish the ideal profile for the patient in question.

3.1 The Mandible

The mandible normally consists of two equal halves, extending from the symphysis up to the condylar and coronoid processes. The symphysis is the fusion line of the two mandibular halves. Each half is called a hemimandible. Its length extends from the symphysis to the articular surface of the condylar process.

The term “parasymphysis” is often used in the USA to describe the area lateral to the symphysis. However, such a term does not exist in anatomical textbooks. Although that term is used to describe a part of the chin of one side it is still not equal to half of the chin promi-

nence. In general we talk about a parasymphyseal area where a specific pathology may be located. But we do not use the term “parasymphysis” in the description of mandibular anomalies nor in orthognathic surgery at all.

The surgeon who has to deal with the many different shapes and positional anomalies of the mandible, while respecting the position of the teeth and the occlusion, will soon recognize that in diagnosis and treatment, one cannot just use the term “mandible” since it consists of several different parts: the horizontal ramus, consisting of the body or base of the mandible and the alveolar process (with the teeth), the chin and the ascending ramus with the condylar and coronoid processes. Their shape and position in relation to each other has to be clearly evaluated in order to make the diagnosis and to plan the proper treatment of the case. Surgery may either move the whole mandible, almost never including the condylar processes, or only parts of it.

3.1.1 Anomalies of the Mandible

I use the term *normomandibulism* to describe a mandible normal in length, width, height and position relative to the other facial skeletal parts. Therefore by the term *retromandibulism* I mean such a mandible in which the horizontal rami are short as if they were shifted into the ascending rami, while in *antemandibulism* they seem to be “pulled out” of the ascending ramus as is done in the sagittal splitting procedure for advancement. The term *micromandibulism* means that the mandible is small in all dimensions, as in the bird face appearance, while in *macromandibulism* the mandible is large in all three dimensions. We talk about *mandibular asymmetry* when the two halves of the mandible are unequal as in hemimandibular hyperplasia or hemimandibular elongation. In *lateromandibulism* the mandible is shifted to one side, both halves being of equal length. Combinations do, of course, occur.

3.1.2 The Horizontal Ramus

The most important part of the mandible is its horizontal ramus. Its shape and position mainly determine the

appearance of the face. It includes the body and the alveolar process. Its vertical dimension, called its height, consists of the body or base of the mandible and the alveolar process, with or without teeth.

Its inferior horizontal dimension, its actual length, extends in the lateral cephalogram from the G-point of the angle to the symphysis (Sy) which is synonymous with the mental protuberance (Pg-point). In order to be able to compare measurements between right and left hemimandibles the cephalograms (lateral and p.a. projection) are useless.

On the panoramic radiograph we will use that Pg-point in the chin area, generally recognizable as a whitish spot, in measurement of the length of the horizontal ramus. The length of the horizontal ramus (HRL) is measured from that Pg-point to the G-point, the most dorsal point (lateral on the OPT) of the angle. Thus we can compare the length of the left and right body of the mandible.

Its superior horizontal dimension is measured from a point in the symphysis region at the level of the apices of the first incisors (B-point) to the curvature in the retromolar region (Rm-point). It is equal to the length of the alveolar process. The inferior length of the horizontal ramus is important for the profile evaluation while its upper length is important for the space available for the teeth. The difference between the upper and inferior lengths varies naturally from one patient to another, depending on the breadth of the ascending ramus. But it can also vary between left and right in the same patient.

The horizontal ramus can be normal in length, or longer or shorter than normal. Its height can be normal or can be smaller or greater (lower or higher than normal). By the term “thickness” of the horizontal ramus we mean the medio-lateral width of the body. It might be normal, or thin (slim) or thick (bulky). By “anterior mandibular height” is meant the full height of the chin including the alveolus in the vertical dimension.

3.1.3 The Mandibular Body or Base

The mandibular body, or base, is defined (Reed and Sheppard 1976) as that part of the horizontal ramus which extends from the lower border of the mandible to the mental foramen. As it is measured as length we need an upper line not just the foramen. In my definition the mandibular body is that part of the horizontal ramus which extends from its inferior border to the upper border of the mandibular canal and its anterior branch. That distance, in the vertical dimension, is called the body height (BH). I define the extent or length of the body from the G-point to the Pg-point. It is equal to the horizontal ramus length (HRL). The alveolus with its teeth sits on top of the mandibular body or base.

3.1.4 The Ascending Ramus

The ascending ramus, often just called the ramus, follows the horizontal ramus dorso-cranially. It begins at the angle of the mandible at its most inferior point (Ag-antegonial point) where in most cases the convexity of the angle usually turns into a slight concavity of the horizontal ramus, and ends at the highest point of the articular surface of the condylar process (HC-highest point of the condyle). That distance is called the height (or length) of the ramus. The ramus can also vary in form and volume and so can the angle which is formed together with the mandibular body.

When discussing with American and British friends the shape and size of the different parts of the mandible, I found great difficulties in expressing myself to their satisfaction in these usages. While the British often talk about the length of the ascending ramus, the Americans call it its height, as we do in the German language. So, ramus height and ramus length are the same. Because of these differences it seems necessary to define strictly the meaning of the different adjectives used.

For measurement reasons the ascending ramus must be divided into several clearly distinguishable parts as in unilateral growth anomalies we want to know in which section of the ramus the abnormal growth takes place. The main part I will call the trunk of the ramus. Its height or length (TL) I define as the distance from the Ag-point to the most inferior point of the semilunar notch (I name it IS-point, derived from its Latin name *incisura semilunaris*). It can be normal or longer (higher) or shorter in relation to its processes or to the body or the other facial skeletal parts.

Its lateral aspect is its breadth or width while its medio-lateral measurement is called the thickness. The width can be normal or wider or narrower than normal. Its thickness can be normal or thicker or thinner or slim.

On top of the trunk are the articular and the coronoid processes. They are not just a part of the ramus. They must be clearly distinguished from the ramus. The horizontal and ascending rami together form the mandibular angle.

3.1.5 The Articular (Condylar) Process

The articular process consists of the condylar head, I will just use the term “condyle”, and the condylar neck. For the latter I will just use the word “neck”. They are diagnostically and from the therapeutic point of view of special interest and importance. The length of the neck (NL) of the condylar process extends from its base up to the base of the condyle, which I define as the insertion

of the condylar capsule. On the radiograph this is not recognizable. As the base of the condylar neck I define a perpendicular line from the IS-point to a tangential line along the neck's posterior surface. Occasionally there is a type of a bend formation between the neck and the trunk. In such a case a line from the IS-point to the middle of that bend may be used to define the base of the neck. The length of the neck may be within average or normal, or it may be short or long or clearly elongated. The neck's antero-posterior dimension is called breadth or width (WN). It may vary from average to thick or slim (thin).

Within the range of normal, the condyle appears in great variations in its size and shape. In spite of its great variations within normal, it is often possible to call a condyle enlarged or reduced in size, in particular in relation to the condyle of the other side.

3.1.6

The Muscle (Coronoid) Process

The muscle process also exists, within the range of normal, in variations of size and forms. Nevertheless, occasionally it is clearly shorter or longer than normal and smaller or larger in its width (antero-posterior dimension) at its base. It extends from its base up to its tip. As the base of the coronoid process (CPB) I define a perpendicular line from the IS-point to a tangential line along its anterior contour. Its end may be normal somewhat or very pointed, or rounded. Its thickness does not need to be described specially.

Clinically, the coronoid process does not play an important role in facial skeletal anomalies, whatever its shape may be. Only cases with remarkable elongation of the coronoid process may cause problems because of the resulting restricted mouth opening. This problem, often wrongly called "coronoid hypertrophy", instead of "coronoid elongation", seems to develop before or during pubertal growth, independent of any other facial skeletal anomaly. If a patient does not have an incisal opening of more than 25 mm, without any obvious joint, muscular or other soft tissue origin, elongation of the coronoid processes must be considered. It is mostly bilateral, but occurs also unilaterally. The diagnosis is simple. When forced mouth opening is absolutely blocked, with restricted opening as mentioned, one has to think of coronoid elongation. The panoramic radiograph will clarify this situation.

The problem is usually solved by cutting through the coronoid processes, without removing them. Postoperative opening exercises are very necessary until normal mandibular opening is secured. These exercises will create scar tissue in a position that will not interfere with the mouth opening.

3.1.7

The Angle of the Mandible

The angle of the mandible is formed by the confluence of the body and the ascending ramus. Both play an important part in its formation. No distinct line, natural or constructed, demarcates the two from each other, nor is such a demarcation necessary. When measuring the length of the ramus or the body we just include the angle area of the other. Anteriorly, the angle starts at the curvature in the retromolar area (Rm-point). Posteriorly, we include visually the G-point.

The angle can vary quite a bit in its shape and angulation. In the average adult, that angle should be about $124^\circ (\pm 6^\circ)$ in the male and $122^\circ (\pm 4^\circ)$ in the female. Therefore, we will talk about a normal angulation or a stretched or pronounced or distinct angle. Its protuberance can be within the normal range, or flat (as in some hemimandibular elongation cases) or protruding (as often found in cases with masseter hypertrophy). The medio-lateral dimension of the angle area may be thick or thin or normal.

3.1.8

The Chin and Its Anomalies

Although the chin does not seem to have its own embryogenetic development, independent of the base of the mandible, it can be variable in its form and consequently deserves independent special corrective surgery. It is located in front of the mandibular base. I determine it by a vertical line drawn from the Frankfort plane to point B (as defined on the lateral cephalogram) extending to the inferior border of the horizontal ramus. From point B downwards we call it the height of the chin and from that vertical line forwards or backwards it is called the prominence of the chin. The chin extends laterally from one mental foramen area to the other.

As mentioned previously, *chin anomalies* have to be evaluated independently. *Normogenia* refers to a chin prominence that is adequate in height, projection and width in relation to the mandible. In *antegenia* such a chin prominence is too far forward, while in *retrogenia* the prominence is too far back. In *macrogenia* the chin prominence is too large in all three dimensions and in *microgenia* too small. In *laterogenia* the prominence is shifted to one side. *Hypergenia* and *hypogenia* describe a chin prominence which is too high (deep) or not high enough (shallow) in the vertical dimension but otherwise normal in shape, width and position. Unfortunately, the chin prominence does not depend on its skeletal elements alone. In addition, variations may exist in soft tissue thickness in the chin region, which require either

a reduction or increase in its thickness or even a displacement. Combinations of the various skeletal elements and soft tissue configurations from normal to abnormal also exist.

3.1.9

Measuring the Mandible

In asymmetrical mandibular growth anomalies we must be able to compare every section of the two hemi-mandibles in order to diagnose where the unilateral anomaly becomes abnormal compared with the other side. The various sections of the mandible have to be clearly defined for determining which part is mainly influenced by the abnormal growth, and to what extent.

The panoramic view is the best routinely taken radiograph which permits comparison of the two halves of the mandible. Radiographs should not be used to evaluate cases when distortions, due to technical origins, are noted.

I am fully aware that some radiographs will not allow all necessary measurement points to be determined precisely, particularly for the articular process. As we know that mandibular asymmetry due to misregulation of condylar growth activity has its origin located close to the surface of the condyle, we are very interested to learn how the individual condyle reacts in its size and shape due to the hyperactivity of one or the other or both condylar growth regulators. As it is not possible to determine the base of the condyle on the radiograph, only the length of the whole condylar process and the width (anterio-posterior extent) of the condyle and its neck can be measured. Accurate measurements of the length and width of the various parts can be obtained on tracings of high-quality panoramic radiographs. For this, measuring points generally known in the evaluation of the lateral cephalogram can be used along with the addition of new measuring points (Fig. 4).

Measurement Points

- | | |
|-----|--|
| G: | gonion point, the most dorsal point of the angle |
| Pg: | pogonion point, equivalent to the mental protuberance, in most radiographs visible as a whitish spot |
| B: | point in the symphysis region at the level of the apices of the first inferior incisors |
| Sy: | symphyseal point, most inferior point of the symphysis just below the Pg |
| Ag: | antegonion point, most inferior point of the angle |
| HC: | height of condyle point, most cranial point of the condylar surface |
| IS: | incisura semilunaris point, the most inferior point of the semilunar notch |
| CP: | coronoid process point, tip of the coronoid process |
| AL: | alveolar point, the highest point of the alveolar crest |
| Rm: | retromolar point, anterior begin of the angle of the mandible |

Measurable Lengths

- HRL: horizontal ramus length, from pogonion to gonion point
- HRH: horizontal ramus height, from inferior border to the crest top of the alveolus. One has to define in which area the measurement was taken
- BH: body height, from inferior border to cranial line of the mandibular canal
- AH: alveolus height, from cranial line of mandibular canal to crest top of the alveolus. One has to define in which area the measurement was taken
- ARL: ascending ramus length, from Ag-point to HC-point (highest point of condyle)
- TL: length of trunk of the ascending ramus, from Ag- to IS-point

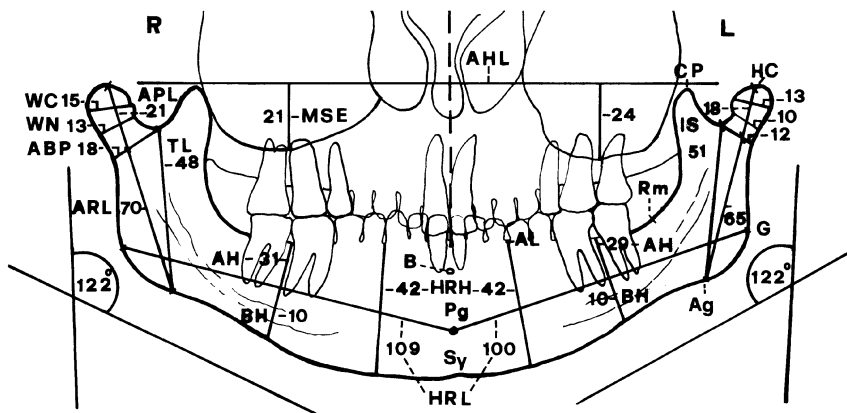


Fig. 4. On the tracing of a panoramic radiograph all wanted measurement points and measurable lengths are shown

APL:	articular process length, from base of neck to HC-point
APB:	articular process base, from IS-point to the most anterior point of the posterior ramus border
NB:	neck base, equal to APB
NL:	neck length, from NB to base of condyle
WN:	width of condylar neck, the smallest distance between the anterior and posterior surface of the neck
WC:	width of condyle, the largest antero-posterior distance of the condyle
CPB:	coronoid process base, a perpendicular line from IS-point to a line tangential to the anterior ramus rim
CPL:	coronoid process length, from CPB- to CP-point
AHL:	articular fossa height line, line between the height of the two articular fossae
MSE:	maxillary sinus extension, most inferior extension of the maxillary sinus from AHL

The angle on the affected side can change greatly, owing to condylar hyperactivity. Therefore the non-affected side should be compared with the affected one. The gonial angle can be accurately measured only on the lateral cephalogram or on good sagittal tomograms. The lateral cephalogram is of no use for comparing the left with the right side, except when the left and right horizontal and ascending rami can be very clearly distinguished. Knowing that the size of the angle on the panoramic view does not permit any conclusion regarding its actual measurement, comparison of the left and right sides can be done on that radiograph.

In some cases, in addition to the panoramic radiograph, good TMJ-ramus tomograms and lateral cephalograms allow the two sides to be clearly distinguished. Unexpectedly large differences have been found in measuring the angles on the three radiographic projections. The measurements on the lateral cephalogram and on the TMJ-ramus tomogram were most likely to be similar, but the measurements of the angles on the panoramic view could differ by as much as 10° in comparison with the two other possibilities. The most accurate measurement is achieved when good TMJ-ramus tomograms are used, as long as enough length of the horizontal ramus can be seen on the roentgenogram.

3.2 The Maxilla

In the maxilla, only its base can be differentiated from the alveolus (with or without teeth). The assessment of the base of the maxilla can only be made in its relationship to the mandible and the other parts of the facial skeleton. The assessment of the alveolar process must be made solely in relationship to its relevant base of the maxilla.

In treatment, it is very important to differentiate between the base of the maxilla and its alveolar process, as the base can hardly be influenced in its size and shape by orthodontic therapy. The opposite is true for the alveolar process. The orthodontist decides where he wants it to be. In patients with extreme overexpansion, the alveolar process may even be beyond the actual base of the jaw.

3.2.1 The Maxillary Base and Its Anomalies

The two palatal bones together make the base of the maxilla. Its thickness is normally around 2 mm only, but I have also found it up to 5 mm and more in pathological conditions (cases of craniostenosis, facial hemihypertrophy, facial osteopetrosis, fibrous dysplasia, Cooley's anaemia and others).

Normomaxillism is a maxillary base which is normal in length, width and position. *Retromaxillism* is a maxillary base which is normal in length and width but which is too far retroposed. *Antemaxillism* is defined by a maxillary base of normal length and width which is positioned too far forward. In *micromaxillism* the maxillary base is too small in length and width. In most cases it also lies too far back and then it may additionally be described by the term *retromaxillism*. However, when we advance such a maxilla forwards it still remains a *micromaxillism* but in a normal position. In *macromaxillism* the maxillary base is too large in all dimensions. *Hypermaxillism* means that the maxilla is too large in height (= vertical maxillary excess). *Hypomaxillism* means the opposite (= vertical maxillary deficiency). However, in both cases it is rather an anomaly of the maxillary alveolus than of the base of the maxilla. *Maxillary asymmetry* means that the maxillary base is asymmetrical in length and width and maybe also in position. The classical example is the maxilla in a severe, hemifacial microsomia deformity. *Lateromaxillism* means that a maxillary base of normal size and width is shifted laterally, while in *maxillary rotation* it is rotated with or without any other anomalies of the maxillary base. For each the most common cause is trauma or in some situations the anomaly occurs after a forceps delivery. Combinations of all these anomalies of the maxillary base may occur.

3.3 The Alveolar Processes

The alveolar processes of the mandible and the maxilla evolve with the development and eruption of the contained tooth germs. Although it sits on the base of the jaws the alveolar process has the capacity to exist independent of a mandibular base underneath it. This is occasionally seen in congenital mandibular defects as in

cases of hemifacial microsomia (see Fig. 18g). It is also seen in those cases in which the base of the mandible has been lost due to surgery or trauma, while the alveolar process containing the teeth remained unchanged for years.

Even in situations without an obvious cause, the alveolar process does not always develop in the same positional relationship to the base of the jaws. This finding is proof of the alveolar process being a separate type of bone structure. It alone allows us to understand why the alveolar process occasionally develops too far forward, distally, laterally or medially and in different shapes.

The positional anomalies of the alveolar processes may develop spontaneously or arise through orthodontic, surgical or other influences. Within the alveolus, the teeth can be in correct angulation or be tilted relative to the pertinent base of the jaw. There can be too little or too much space between them or they may be just perfectly in alignment.

3.3.1 Anomalies of the Alveolar Processes

For treatment, alveolar anomalies have to be diagnosed separately and named accordingly, independently of the anomalies of the bases of the mandible and the maxilla. *Normoalveolism* is an alveolar process normal in height, width and position relative to the respective base of the jaw. In *antealveolism*, an alveolus normal in size and width is positioned forward on the base of that jaw, and in *retroalveolism* the alveolus is situated too far posteriorly. These anomalies are not infrequently found in the mandible. In *macroalveolism* and *microalveolism*, the alveolar process is either too large or too small in all dimensions and in *hyper-* and *hypoalveolism* an alveolar process in normal position is either too high or too low in the vertical dimension. We also speak of *latero-* and *mesioalveolism*, when the alveolus is shifted laterally on the base of the jaw, or mesially. Tilting of the alveolus has to be described separately. Since these anomalies can occur in both the mandible and in the maxilla, in the whole alveolar arch or only locally, we will have to add to the description of the existing anomaly of the alveolar process the words maxillary or mandibular, complete or local. Combinations also exist.

3.4 Abnormal Angulations

It is obvious that the angulation between the various components of the facial skeleton, in particular the jaws, affects the external appearance as well as the occlusion. It does not seem useful to recommend a separate terminology for these variations also. They are made sufficiently clear by standard cephalometric evaluation.

3.5 Long and Short Face

The full picture of an anomaly may not be adequately described by the description of the anomalies of the various parts of the facial skeleton alone. In addition to describing anomalies in the sagittal and horizontal planes, it is also necessary to describe and diagnose anomalies in their vertical dimension. Whereas vertical anomalies of the alveolar processes and of the chin are adequately described by terms (*hyper-* and *hypoalveolism* or *-genia*), precise terminology is required for the vertical discrepancy that exists among the bases of the facial thirds. These are the long and the short face anomalies and the open bite cases.

In the American literature, the terms *long-face syndrome* and *short-face syndrome* are often used. There is no justification for naming these facial anomalies syndromes. In medical terminology a syndrome is either a disease or an anomaly that involves a number of signs and symptoms and of which at least several signs or symptoms have to be present in order to call the disease or the anomaly a syndrome. This definition is not true for either the long-face or the short-face syndrome. The only consistently present sign is either the vertical shortness of the face or its excessive height. But one sign alone does not constitute a syndrome. Therefore, these anomalies should be just named either long-face or short-face. In addition, the origin of the long- or short-face has to be added. It may be due to the maxilla or the mandible or both. Therefore, the condition should be termed *maxillary* or *a mandibular* or *a bimaxillary short or long face*. It may be that the anomaly is due to a vertical anomaly of the alveolar processes only or of the position or angulation of the bases of the jaws or of the height of the prominence of the chin. Another cause may be the vertical bite relationship as in the deep overbite and the anterior open bite.

A *maxillary long face* is either a *maxillary basal long face* (with the base of it positioned too low) or a *maxillary hyperalveolism long face* (when the alveolus is too high) or both. The *maxillary short face* will be the reverse. The *mandibular long face* is either a *mandibular basal long face* (with the base of the mandible positioned too low) or a *mandibular hyperalveolism long face* (when the alveolus is too high) or both. The *mandibular short face* will be the reverse.

3.6 Open Bite Anomalies

The open bite may be due to an anomaly of either the maxilla or mandible or both, basal or alveolar. Almost every open bite situation produces a long face, at least the so-called anterior and so-called total open bite. *Anterior open bite* refers to an open bite that is limited to as

far back as the premolar regions only. The *maxillary anterior open bite* may be due to a position of the anterior maxillary segment that is too high or be due to a position of the posterior maxillary segments that is too low. An anterior maxillary hypoalveolism can also be the cause. In all three situations, an overexaggerated curve of Spee is present.

The *mandibular anterior open bite* can be alveolar or basal, or both.

Total or circular open bite refers to occlusion of the last molars only (uni- or bilaterally). This situation again may be due to an aberrant position of the maxillary or mandibular base only, or of both. And even the height of the alveolar processes may be the cause.

In addition, *lateral open bite* only or *posterior open bite* only must be distinguished. The open bite may be uni- or bilateral and may arise in the maxilla or the mandible, or both. Most of these open bite problems are due to hypoalveolism or contralateral hyperalveolism.

3.7 Conclusion

Using the terminology suggested in this chapter should allow more efficient communication among treatment providers and would direct ourselves primarily towards more accurate diagnosis and more effective treatment.

Documentation for Diagnosis, Treatment Planning and Follow-up

4.1

Fundamentals Regarding Case Documentation

Case documentation serves three purposes: for diagnosis, treatment planning and follow-up. Evidently, these purposes require comparable and standardized documentation. These data are: standardized photographs and radiographs and models of the teeth. In cases of growth anomalies, an additional scintigraphic investigation of the condyles is necessary. This is of special importance in almost all cases of abnormal mandibular growth to ascertain if the abnormal growth has to be arrested by a high condylectomy or whether growth will continue during and after orthodontic or surgical treatment. My experience tells me: *“Too early corrective surgery on the maxilla will result in reduction of its growth capacity and too early osteotomies of the mandible will end up with relapse”* (Principle No. 28) and *“Growth can destroy your early good result”* (Principle No. 27).

4.2

Who Provides These Documentation Data?

As most of these cases present a multidisciplinary problem each speciality involved is interested to have these data made from that particular speciality's point of view. Evidently, these data should be the same for all of them, particularly for the orthodontist and surgeon. This is true for the clinical investigation data as well as for the photographs and radiographs. For this reason orthodontists and surgeons have to come to an agreement on this problem.

In those cases which present with speech problems, the speech therapist must also be consulted. The general medical condition of the patient may require investigation by a paediatrician or internal medicine specialist or endocrinologist in a case of questionable acromegaly or non-union after osteotomy. When the anomaly or its therapy involves the middle or the upper third of the face, the ENT specialist and the ophthalmologist must also be consulted.

All these pretherapeutic investigations are necessary, not only from the point of view of diagnosis and treatment planning, but also for medico-legal reasons. There-

fore it is very important to have the patient's informed consent to use pre- and postoperative photographs for teaching and publication purposes. So we have on our registration forms which are signed by the patient, the following wording: I agree to the use of material from my records for teaching purposes so long as my name is not revealed.

4.3

Clinical Investigations

Clinical investigation should be standardized in order not to forget anything. The most important part is what you can see with your eyes. That means first of all *“Learn to see, not only to look at”* (Principle No. 43). Therefore *“Pray every day to God that you do not become blind with open eyes”* (L. Obwegeser sen.) (Principle No. 42) as my father has taught us children. Because *“What you do not have in your brain your hands cannot do”* (Principle No. 46), a principle which I often told to my students.

The clinical investigation includes:

- The patient's complaints, when they started and his wishes and desires
- The patient's history, when did the anomaly start and any possible cause
- The family history should be inquired into very precisely, whether the same anomaly, a similar one or another one has been observed
- The patient's general medical condition
- Extraoral and intraoral inspection and palpation with clear description of the anomaly and the other facial parts
- Examination of the extraoral musculature and tongue and their function, of the oral mucosa, of the salivary glands and of the function of the facial nerves
- TMJ palpation and auscultation findings
- Movement of the mandible in all directions
- Whenever one is in doubt about facial symmetry follow the principle: *“When you are not sure whether a facial asymmetry exists or not, place the patient's head in the upright position, then draw a horizontal line above the eyebrows and to it an imaginary perpendicular line from the middle of the forehead. Then it can be seen clearly whether a facial asymmetry ex-*



Fig. 5a,b. Evaluation of facial symmetry and asymmetry (see case Fig. 50). **a** Looking at the patient's face from in front the nasal bridge seems to deviate to the right while the chin seems to be almost in the facial midline **b** with the horizontal line drawn above the eyebrows and a perpendicular line to it in the middle of the forehead the differences between the two sides become very evident

ists or not and what is on which side of the face (nose, chin, teeth midline) and what the differences are between the two halves of the face” (Principle No. 16) (see Fig. 5, Fig. 33g and Fig. 50h)

- Occlusion and occlusal plane including insertion of a wooden spatula between the teeth in the molar region in the closed teeth position to observe any canting
- Detailed teeth situation. Many surgeons do not bother about “such dental problems”. Such an opinion should be decried not only for treatment planning reasons but also for avoiding surgical complications and for medico-legal reasons.

4.4

Radiographic Documentation

In order to diagnose a mandibular anomaly and its relation to the maxilla it is not often that one X-ray projection alone is sufficient. The following projections are needed in most cases:

Panoramic View of the Mandible in the Closed Teeth Position (Central Occlusion). It is the best radiograph for any mandibular anomaly, independent of its clinical presentation and its aetiology. In the closed position the anatomical shape of the mandible and its structure is more easily recognizable than when the panoramic radiograph is taken in a slightly open position of the mandible. The projection in the closed position permits clear diagnosis of important points: generally, when the maxilla is not involved in a posttraumatic or growth anomaly, the nasal septum and the nasal crest of the pal-

atine bones indicate the facial midline. That facial midline is very important from many points of view: the position of the midline of the upper and lower teeth, the chin prominence and the symphysis and, of course, the overall symmetry or asymmetry of the mandible. It gives a clear view of the shape of the mandible and of all its sections, including the position of the mandibular canal as well as the shape, size and some indication of the state of its health. It also permits evaluation of the structure of the mandible. Only the uppermost part of the condyles is not delineated properly. When the panoramic projection is taken in a slightly open position of the mandible, as is usually done for dental purposes, then an asymmetry of the chin and the lower teeth midline may not be as clearly visible or it may even be corrected by the mouth opening.

However, this projection is also the best one for identifying any pathology or impacted or supernumerary teeth within the mandible which might cause some problems for both the orthodontist as well as the surgeon.

Mandibular p.a. Projection in the Maximum Mouth Opening Position. I always want to compare the length of the two ascending rami and their width. The latter is particularly helpful to the surgeon. He wants to know how thick the ramus is for a splitting procedure. However, one must be aware that in the H.H. and hybrid form cases, the thickness of the two rami are not comparable in this projection as the hyperactive side pushes the other side laterally and rotates it. This projection also shows the trabecular pattern of the mandible and the condyles.

Lateral Cephalometric Radiograph. This is requested almost routinely by both the orthodontist and the surgeon. It gives important information about the bony framework and any possible difference in height between the two sides and its influence on the patient's profile.

P.a. Cephalogram. This is often requested and is helpful in the mandibular asymmetry cases.

TMJ Sagittal Tomograms Including the Ramus and Angle Area. This projection is almost mandatory for diagnosing abnormalities of the condyles and their necks as well as for comparing the two ramus-angle areas. This projection may sometimes be valuable in both closed and maximum opening positions. Normally, in the closed position it gives us all we want to see. Naturally, computerized tomograms are the best, but they are more expensive and require more radiation exposure than conventional tomograms.

Paranasal Sinuses, Water's Projection. I also like to have this in every case. It gives information about the position of the crista galli thus telling us where the facial midline really is and whether the nasal septum and the nasal crest of the palatine bones and the midline of the upper teeth are in the facial midline. Furthermore that projection shows us whether both maxillary sinuses are of equal size and lets us compare the height of the zygomatic alveolar crest on the two sides, indicating whether there is an increase or reduction on one side. Furthermore it gives very good information about possible pathology in the maxilla and maxillary sinuses.

Special Dental Radiographs. These are mandatory for all non-vital teeth in order to ascertain whether there is any pocket formation or apical infection. Both may influence the orthodontic as well as the surgical treatment. A vitality test on all teeth is also indicated.

Hand and Wrist Radiograph. As is generally known and also proven by some of the cases to be discussed, the skeletal age is not always congruent with the chronological age of the patient. The patient's height also does not disclose how much additional growth of the facial skeleton is still to be expected. On the basis of the hand and wrist X-ray, one can clearly define the skeletal age of the patient and can tell what the final size of the patient is going to be (W. Greulich and S. Pyle 1959).

The criteria are the amount of ossification of the epiphyseal plates of the radius and ulna and of the metacarpals. Therefore the wrist radiograph can give very valuable information to the orthodontist and the surgeon. This is particularly true when comparing with the scintigraphic findings.

Special Radiographic Investigations. These may be required in special cases in which mandibular growth anomalies are only a part of a more generalised facial or even general skeletal anomaly.

Carotid or Superselective Angiography. This may be required and is almost a necessity in all cases of maxillo-mandibular asymmetries of uncertain origin (see cases of Fig. 20).

Scintigraphy. This investigation, for evaluation of the ongoing cellular activity in the condyles, is essential in most cases of mandibular growth anomalies. This important investigation will be discussed in a special chapter.

Three-Dimensional Imaging of the Facial Skeleton with Its Soft Tissues. Computerized techniques have been developed recently, permitting production of three-dimensional radiographic pictures of the facial skeleton and its investing soft tissues (S. Arridge et al. 1985). Computed tomography of the facial skeleton is the basis of this system. The soft tissue data are gained by scanning the surface with a laser. Information gained enables the computer to generate a three-dimensional picture. The advantage of this computerized method is the possibility of storing a large amount of data thus enabling us to track growth as well as changes in soft tissue and skeletal contour. It also can assist the planning of orthognathic surgery three-dimensionally. For diagnosis and treatment planning of facial skeletal anomalies it is mainly the three-dimensional imaging of the facial skeleton which is important.

Magnetic Resonance Imaging. This examination does not involve any radiation exposure. However, it is much better for evaluating soft tissues than the skeleton. Therefore, it does not have any real indication in the field of mandibular growth anomalies except for desired information on the mandibular joint disc and other soft tissues.

4.5 Photographic Documentation

The photographic documentation must also be standardized. There are a lot of projections taken which are not always necessary. In order to minimize the possibility of forgetting one or other projection and yet not to take too many, we use a request form (see below) which is applicable to both the orthodontist and the surgeon. When referring the patient for photographs the desired projections are underlined. This request form is also helpful if one takes the photographs in one's own office.

Standardization is essential for comparison of pre- and post-treatment photographs for result evaluation. For this purpose every photograph is taken with the pa-

tient in a defined position. This is particularly necessary for photographs of the patient's face either in the lateral, semilateral or front view. For all photographs, the patient is seated on a type of chair where he can sit firmly and which gives the photographer the possibility of moving the patient up and down and to rotate him so as to have him always at the correct height for the camera. An old dental type or a hairdresser's chair with the possibility to have the head resting steadily is better than an ordinary chair with a revolvable seat. Also the distance of the patient's face from the camera must always be the same whether the patient is an adult or a child. For these

views, the patient sits in an upright position and looks at a fixed point (red or yellow) on the wall. The photographer will raise the patient's chair exactly to that height so that his eyes are at the same height as the fixed point on the wall which should be about 2.5 m distant. A stick with a mark helps to guarantee that the patient's eyes are at the same height as the red point on the wall. When the patient looks at that point and the camera is always at the same distance, comparable photographs will always be obtained.

The camera should be easily movable in any direction, but always fixable in the same position for the



Fig. 6a,b. Arrangement for achieving standardized photographs. **a** The patient sits in an old dental chair with the head resting on the head support, fixing with his eyes the red point on the opposite wall. The mark on the stick measures the height of the eyes equal to the height of the red point on the wall. **b** For the profile photograph, the chair is rotated 90°. In this position the patient now sits in front of a blue wall, again fixing his eyes on another red point at the same height

same type of photograph. We used a scissor-like support from an old X-ray machine. It is fixed to the wall (Fig. 6a). In the direction of the camera, behind the patient, the same background is always used. I prefer a large blue sheet (Fig. 6b).

The following request form indicates what projection(s) are wanted:

4.6 Request Form for Photographs

New/old	
Surname	Photo-file No.
First name	Pat. record No.
Date of birth	Diagnosis:
Face, front view	Resting position – with max. mouth opening – with spatula – forehead wrinkling and lips pouting – smiling
Profile view	Left – right – smiling
Semiprofile view	Left – right – smiling
Face	From below – from above
Lower facial half	Resting position – lips pouting – showing teeth
Columella and tip of nose	From below – lateral – lateral oblique
Eyes	Looking forwards – upwards – downwards – to left – to right – closed
Occlusion	Front – left – right, from above – from below – with and without denture(s)
Maxilla	Full view – mirror view from below
Mandible Vestibulum	Full view – mirror view from above
	left – right – maxilla – mandible
Cheek inside	Left – right
Soft palate	Pharyngeal wall
Tongue	Resting – protruded
Special projections	
Film No.	Date of request: Requested by:

4.7 Plaster of Paris Models of the Teeth

In all cases of occlusal involvement, I generally had three sets of models of the teeth made:

- 1. One pair of stone models with a wax bite.
- 2. Two pairs of (soft) plaster of Paris models for planning work. They are used to perform a model operation. When the maxilla also has to be moved they must be fixed in an articulator according to the bite registration.

The pair of stone models is a study model of the pre-therapeutic situation. The soft plaster of Paris pairs can be used for several purposes: to set up the teeth in the desired position; model operation; preparation of an acrylic splint.

4.8 Facial Mask Model of Plaster of Paris

Such model masks are occasionally helpful for teaching purposes as well as an aid to surgical planning. In cases of facial asymmetry, in particular of the zygomas, forehead or ramus-body hypoplasia such a mask is very useful in determining the amount of material (cartilage, silastic, etc.) needed for facial symmetrization.

To make a facial mask, the whole face including the eyelids is covered with vaseline. Then a cardboard halo is placed around the face in front of the ears. Next, a soft lubricated rubber or silastic tube is inserted on each side of the nose after the nasal cavities have been anaesthetized. Then a soft, elastic impression material is poured over the face, the nasal tubes protruding through it. When the impression material has set, its free surface is hardened by adding a layer of fast drying plaster of Paris. As soon as this is set, the whole impression can be removed without distorting it. It is then filled with plaster of Paris, thus producing a facial mask.

4.9 Stereolithographic Reproduction of the Facial Skeleton

When I became more deeply interested in the fascinating field of facial skeletal anomalies, I started to collect or copy skulls of all the facial skeletal anomalies I could find. My then coworker, H.P. Freihofer, went to several institutes of anatomy, anthropology and pathology to find such skulls. They were lent to me temporarily for radio- and photographic documentation and for copy-



Fig. 7. Skull copy, in acrylic, of a patient with a severe hemifacial microsomia

ing. The chief dental technician of my clinic, Mrs. E. Bai, took impressions of the various parts of the skulls and poured them in acrylic of natural bone colour. The results were phenomenal. These skull copies were so realistic that when I took such a copy to my very good friend Dr. Paul Tessier he thought it was an original skull. He raised the vault of the cranium and had a look at the skull base. He said we should be able to copy these interesting skulls of cranio-facial anomaly patients (see Fig. 7). He could hardly believe that he was holding a copy in his hands. They are helpful indeed for understanding and for treatment planning of cranio-facial anomalies.

Today we have the possibility of producing an accurate copy of the patient's skull, not only of a cadaver skull (C. Hull 1986). These skull reproductions are of great clinical and technical value. Whether this possibility is going to be used frequently in cases of mandibular growth anomalies will depend on the specialists consulted as it is also an expensive form of documentation. I personally would like to have reproductions of all types of mandibular growth anomalies, particularly that of a very severe case of a unilateral hybrid form which had started early in childhood. These reproductions are not as natural to look at as the copies of the cadaver skulls. However, they are quicker to produce and probably more precise and less expensive.

Scintigraphy, a Diagnostic and Treatment Planning Aid

5.1

Background Knowledge

Radionuclides are commonly used for diagnostic as well as therapeutic purposes. Their diagnostic use is called radionuclide imaging or scintiscanning or just scintigraphy. Technetium-99m-methylenediphosphate (Tc-99m-MDP) is the most commonly used radiopharmaceutical substance for obtaining information about cellular activity within the bones. For the facial skeleton, the use of bone-seeking radiopharmaceutical uptake in the assessment of facial growth and development was first investigated by G. Cisneros (1982).

Almost all our cases (tumours, osteomyelitis, growth abnormalities, vascular abnormalities, etc.) have been referred for scintigraphic evaluation of the facial skeleton to the Clinic and Policlinic for Nuclear Medicine (Director Prof. Gustav K. von Schulthess) of the University Hospital, Zürich. On average 500–600 MBq (Megabecquerel) Tc-99m-DPD (3,3-diphosphono-1,2-propanedicarboxylic acid) was injected intravenously and 2–4 h later, head scans were taken. Three dimensional data are acquired using single photon emission computerized tomography, called SPECT. From the acquired raw data an axial (transverse) SPECT, a coronal SPECT and a sagittal SPECT images are counted (Fig. 8a–c). Also plane scans can provide sufficient qualitative information but not quantitative measurements in relation to a normal or contralateral part. The result of the scintigraphic investigation can be produced on laser prints or impressively on coloured paper to facilitate patient information (Fig. 8d).

Bone scanning is highly sensitive but non-specific: when clear hyperactivity is found, it does not tell us whether it is due to inflammation, malignancy, hypervascularization, reparative process or growth activity. The findings have to match the clinical picture. This is valid for all bone scanning, including the facial skeleton.

Before any diagnosis can be made, the average normal scintigraphic pattern of the bone in question must be known. This is particularly important when scintigraphy is used for diagnosis and treatment planning of mandibular growth anomalies. Therefore, the measurable ratio of uptake (RU) of the radiopharmaceutical substance can give quantitative information as to

whether both sides of the mandible, in particular the condyles, are at the same stage of development or whether there is a difference. In relation to our topic, a difference can be due to six possibilities:

1. Unilateral hyperactivity compared with contralateral isoactivity
2. Unilateral hyperactivity compared with contralateral hypoactivity
3. Bilateral asymmetric hypoactivity
4. Unilateral normactivity compared with contralateral hypoactivity
5. Bilateral normactivity when mandibular growth is not finished
6. Unilateral hyperactivity due to any other pathology

In the growing patient, the diagnosis is often not all that easy. The scintigraphic findings are exact data. Their proper evaluation relies on the referring physician, especially if there is no possibility of a comparison with what is normal for that patient's age. A wrist radiograph may indicate whether or not the general skeletal growth has already ceased. That can clearly help, in some cases, to interpret the scintigraphic findings correctly, at least when growth has ceased. During the normal growth period, correct interpretation of the scintigraphic findings in the condylar region may still be uncertain. I have experienced that, finally, the histology of the resected condyle revealed whether a long condylar neck was due to H.E. or the patient's normal growth pattern (see case of Fig. 51).

For this reason it seems very important to have clear images and data regarding condylar growth activity measured by scintigraphy in the various age groups of skeletal growth.

There is very little published on scintigraphic findings in the mandible in the various age groups. L. Kaban et al. (1982) have tried to ascertain the extent of uptake of Tc-99m-MPD found on average in 6 age groups ranging from 1 to over 20 years old. They compared the ratio of uptake (RU) in the condylar region with that in the fourth lumbar vertebra. Their findings were based on a group of 34 patients. Naturally, the number of cases investigated is very small, almost negligible, in some of their six groups. In 1995 L. Kaban et al. published the results of 90 cases, also in 6 age groups, from 0–30 years

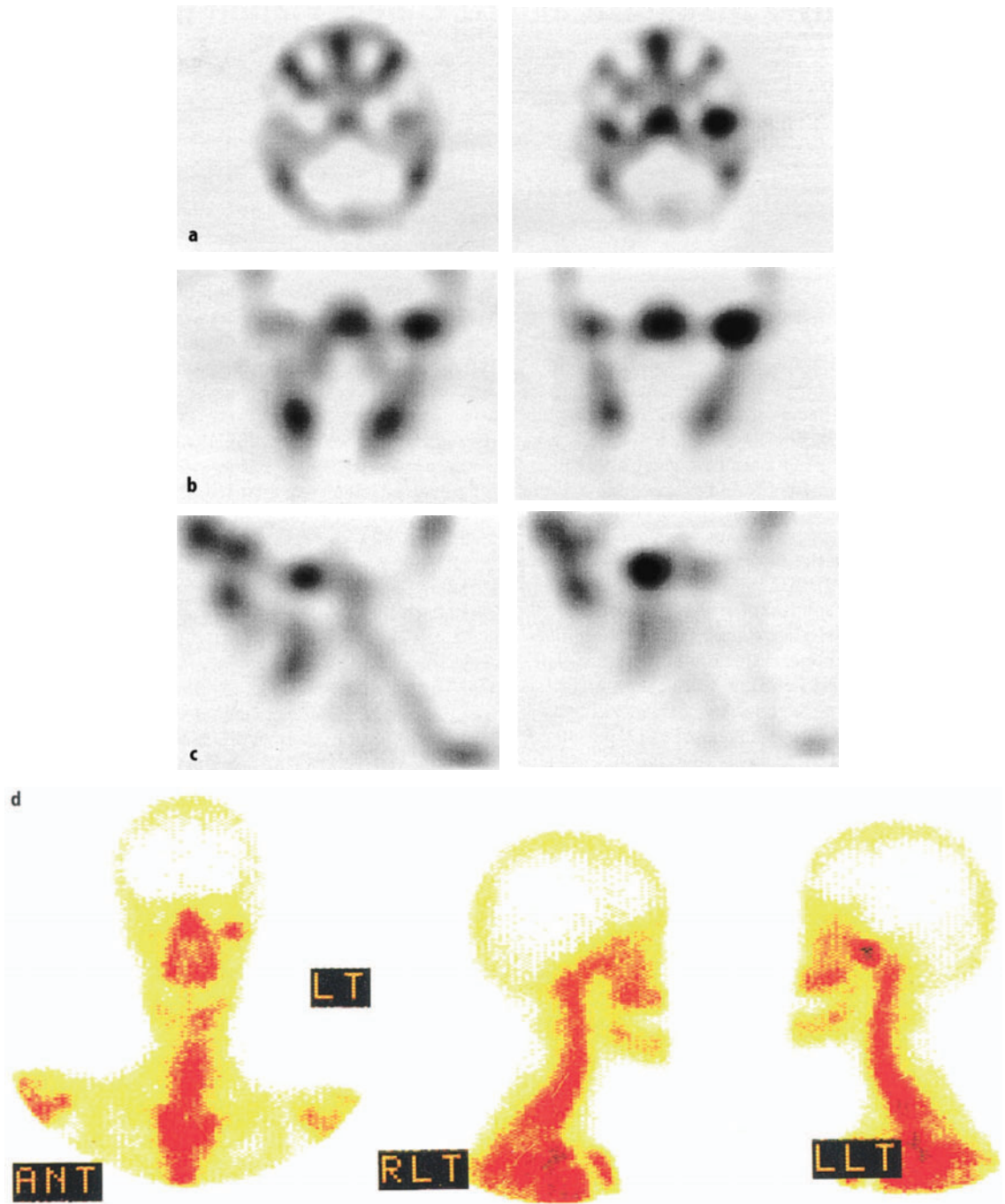


Fig. 8a–d. Scintigraphy of the TMJ. SPECTS in the three different axes. **a** axial/transverse (top), **b** coronal (middle), **c** sagittal (inferior). **d** Skull and neck in p.a. and lateral view projection in colour for easier explanation to the patient

Age	Ratio
0-2	2.0-1.85
2-5	1.85-1.65
5-10	1.65-1.30
10-15	1.30-1.10
15-20	1.10-0.70
20	<0.70

Fig. 9. Approximate RU by age, taken from linear regression analysis [with permission from and thanks to L. Kaban et al. (1995)]

old. The RU declines from each younger group to the older. They found the same results as G. Cisneros and L. Kaban (1984) had found in 21 patients (Fig. 9).

Although the number of cases included in each group is not mentioned, the results seem to represent useful data which permit the investigator to compare the findings in the patient referred to him, with the normal values. It is also very useful for the nuclear physician to know that any difference in activity of radioisotope uptake between the left and right condyle, greater than 10% (M. Pogrel 1985) or 12% (P. Robinson et al. 1990), appears to be significantly abnormal.

Computerized skeletal scintigraphy for assessment of mandibular asymmetry (G. Cisneros and L. Kaban 1984) is able to determine whether or not some growth activity is still going on. If not, then normal mandibular growth may have ceased or there is lack of growth activity due to congenital or aquired condylar hypoactivity. As long as there is more than 0.70 RU condylar activity going on, one should either postpone surgical treatment or eliminate the activity in accordance with the author's principles "*Eliminate the cause of excessive growth as early as possible*" (Principle No. 30) and "*If you do not remove the persisting cause of a facial skeletal anomaly, relapse of your corrective surgery will be preprogrammed*" (Principle No. 31) since "*The active cause of a facial skeletal anomaly is stronger than screws and plates. Plates and screws cannot prevent a relapse if the cause of the anomaly is still active*" (Principle No. 32). Therefore, "*The cause of a given anomaly should either be inactive or eliminated prior to definitive surgery*" (Principle No. 29).

In a comparison of histological with scintigraphic findings R. Gray et al. (1990) suggested assessing bone scans together with anatomical images, preferably produced by computed tomography to obtain the full benefit from radioisotope imaging. They concluded that visual examination of bone scans by experienced observers is a valid means of assessing bone activity in condylar hyperplasia.

5.2 Clinical Consequences

Before bone scintigraphy was available for evaluating the condylar growth situation, we "walked in the dark" as seen in case Fig. 28. Due to that case, we learned and could prove the influence of condylar growth hyperactivity on the ipsilateral hemimandible. In 1961, scintigraphy did not exist as a valuable diagnostic aid in finding the cause of a severe relapse after corrective mandibular osteotomies. Since then, we knew that we had either to resect the condyle of a mandibular growth asymmetry or to wait until growth had ceased. The latter was the method of choice for the slowly growing mandibular asymmetries. Photographs, models and radiographs were compared 6–12 months after the patient was seen initially in order to find out whether or not there was still abnormal growth activity. If there was still some change and continued abnormal growth of the mandibular half, we waited and reexamined the patient again 6 months later. Only when growth had finished could we plan the surgical correction. Later we learned that some of our patients reported a standstill of the asymmetrical growth and yet we found by scintigraphy still ongoing hyperactivity (see case Fig. 32).

For the orthodontist too, not just for the surgeon, the most exact knowledge regarding the degree of condylar activity is important. Even in cases of symmetrical growth or positional abnormalities of the mandible, the orthodontist wants to know what amount of condylar growth activity exists in the particular case for treatment planning and prognosis. This is true for cases of retromandibulism as well as antemandibulism. He cannot expect to advance orthodontically a retruded mandible in a 14-year-old child when mandibular growth has already ceased nor will he be successful in his treatment of an antemandibulism case when severe growth activity in the condyles is still found.

The consequences are that *we have to obtain scintigraphic information* regarding the amount of ongoing growth activity in the condyles *in all cases of*:

1. Mandibular asymmetry due to clearly unilateral growth hyperactivity or hypoactivity.
2. Mandibular asymmetry of uncertain origin
3. Mandibular symmetrical growth deficiency or excess when growth has not yet clearly ceased

This information will help both the orthodontist and the surgeon to plan the treatment properly. This means that, in some cases, the surgical mandibular set back or advancement operation can be performed at the age of 15 or even earlier (see case Fig. 45). But it might also tell us that at the age of 18–19 years relapse can be expected after a setback operation.

Summarizing, I suggest that skeletal scintigraphy should be incorporated in the diagnostic work-up in a great percentage of the cases of maxillo-mandibular disharmony, much more often than it is used today. It must be incorporated in all so-called condylar hyperplasia cases, as the result of scintigraphy may direct the treatment plan (S. Matteson et al. 1985).

The order form should not only ask for scintigraphy of the TMJ condyles to detect the activity relation between left and right, but should ask also specifically for simultaneous scintigraphy of the fourth lumbar vertebra in order to obtain the actual RU figures for comparing the left and

right side. That will tell us whether the ratio is within a normal range for that age. I have been informed that it is technically not possible to gain the RU ratio without specifically comparing the tracer uptake of the fourth lumbar vertebra with the uptake in the condyles. It is obviously not possible to do this retrospectively. For that reason the referring doctor has to ask specifically for this investigation, probably best by informing the nuclear medicine physician of the chapter on "Skeletal Scintigraphy for Assessment of Mandibular Growth and Asymmetry" written by L. Kaban et al. (1995).

Principles in Treatment Planning of Facial Skeletal Anomalies

In treatment planning considerations of mandibular growth anomalies, one has to separate those of condylar hyper- and hypoactivity cases. I myself have learned from my great teachers Richard Trauner, Sir Harold Gillies and Eduard Schmid, that for every problem in planning and treating we will encounter facts which for teaching purposes are best formulated in clear principles. Only general principles will cover both anomalies and all of their variations.

The most important principles for both specialties, surgeons as well as orthodontists, I learned from the greatest pioneer in facial reconstructive surgery of all time with whom I had the privilege to train: Sir Harold G. Gillies. His two most important principles say: “Patients with a facial anomaly primarily wish to have a normal appearance” (H. Gillies 1952) (Principle No. 1), “First diagnose then treat” (H. Gillies 1952) (Principle No. 2).

In my teaching I have also always tried to make myself and things clear by formulating in principles what I wanted to get across to my students. I had learned these principles through my intensive teaching with my trainees and through the discussions with my friends, Dr. Paul Stöckli, Professor and Head of the Department of Orthodontics and Paedodontics at the Zürich Dental School and with his Associate Professor, Dr. Ulrich Teuscher.

The first time I lectured on these principles in treatment planning for facial skeletal anomalies was in July 1986 at Chapel Hill, under the patronage of Dr. Bill Terry, a good friend and pupil of mine. As that lecture was so well received, I used it again and again, on request, in many countries.

Dr. Ulrich Teuscher, who fully agrees with these principles, and I have conducted several postgraduate courses on this subject based on our mutual agreement on treatment philosophy. We applied these principles in our own mutual cases and we used them to demonstrate their application on cases used in our teaching and lecturing to our audience, both surgeons as well as orthodontists.

Based on a large amount of experience I formulated the following important principles for treatment planning of facial skeletal anomalies. If we adhere to them we cannot go wrong:

- Principle No. 3 “Any reconstructive surgery of the facial skeleton should be performed without incisions on the face”.
- Principle No. 4 “In corrective surgery of the facial skeleton, facial incisions should be forbidden for a maxillofacial surgeon”.
- Principle No. 5 “The facial skeleton is the framework that determines the shape of the face. Therefore in facial reconstruction first the bone then the soft tissues” (H. Pichler 1948).
- Principle No. 6 “The size, form and position of the bases of the facial thirds determine the profile type. Therefore, a real alteration of the facial profile can only be achieved by displacing the bases of the facial thirds”
- Principle No. 7 “In the reconstruction of a facial skeletal abnormality, the main aim must be correct positioning of the various parts of the facial skeleton”.
- Principle No. 8 “In treatment planning for facial anomalies first the profile, then the occlusion”.
- Principle No. 9 “If alterations of the bases of the facial thirds are required, dental alignment and the occlusion must not limit the goal set”.
- Principle No. 10 “When planning the correction of a maxillo-mandibular anomaly, consider the jaws as if they were edentulous. Then you will be able to bring their bases into the proper relationship and not just the teeth only”
- Principle No. 11 “A good occlusion does not automatically make a good profile and vice versa”.
- Principle No. 12 “The profile is not the only goal. Correct occlusion and function and facial symmetry are as important”.
- Principle No. 13 “In profiloplasty, one must distinguish between the need to alter the basic profile type or the need to alter the profile accessories only”.
- Principle No. 14 “The facial soft tissues, the teeth and the nose are only accessories in forming the profile. They influence only the profile line but not the profile type”.

- Principle No. 15 "Rotation of the lower half of the facial skeleton is the key to correction of facial height asymmetry".
- Principle No. 16 "If you are not sure whether or not a facial asymmetry exists, have the patient's head in the upright position, then draw a horizontal line above the eyebrows and to it an imaginary line perpendicular to the middle of the forehead. It can then be seen clearly what is on which side of the face (nose, chin, teeth midline) and what the differences are between the two facial halves".
- Principle No. 17 "In midface hypoplasia, correct the upper half according to the requirements of the eyes and orbits and the lower half according to the occlusion and the need of the vertical height".
- Principle No. 18 "Free bone grafts are like babies. They need to be "kept warm" by closely approximating soft tissues around them".
- Principle No. 19 "Cortical bone shrinks like a mushroom in the sun, and cancellous bone melts away like an icecream when used as onlay grafts for contour correction".
- Principle No. 20 "In orthognathic surgery, the segments must be made so loose that they can be easily overcorrected with a pair of tweezers".
- Principle No. 21 "Orthodontics can help a lot but it can also make the necessary surgery almost impossible".
- Principle No. 22 "In the treatment planning of a facial skeletal anomaly, proper angulation of the teeth to their respective jaw base is primarily more important than any other cephalometric angle".
- Principle No. 23 "The anterior position of the lips depends on the position of the alveolar process. To correct labial inversion advance the alveolar process; for correction of labial ectropion move it backwards".
- Principle No. 24 "Overexpanding the maxillary arch cannot correct retromaxillism, nor can retruding the mandibular teeth correct antemandibulism".
- Principle No. 25 "The goal of presurgical orthodontics must be to:
- arrange the teeth in proper angulation to the jaw base, independent of the number of extractions necessary
 - not to extract teeth when there is enough length of the base of the jaw to place all of them into correct angulation to it
 - create a normal curve of Spee
 - create dental arches which will fit together".
- Principle No. 26 "Inadequate planning may easily lead to a disastrous result, functionally as well as aesthetically".
- Principle No. 27 "Growth can destroy your early good result".
- Principle No. 28 "Too early corrective surgery on the maxilla will result in reduction of its growth capacity and too early osteotomies of the mandible will end up with relapse".
- Principle No. 29 "The cause of a given anomaly should either be inactive or eliminated, prior to correction".
- Principle No. 30 "Eliminate the cause of excessive growth as early as possible".
- Principle No. 31 "If you do not remove the persisting cause of a facial skeletal anomaly, relapse of your corrective surgery is preprogrammed".
- Principle No. 32 "The active cause of a facial skeletal anomaly is stronger than screws and plates. Plates and screws cannot prevent a relapse if the cause of the anomaly is still active".
- Principle No. 33 "In the correction of facial skeletal anomalies the surgeon's eyes, experience and intuition are more important than tracings and computer planning".
- Principle No. 34 "Treatment planning of facial skeletal anomalies is an intellectual product based on fundamental knowledge of the subject, experience, imagination and intuition. Executing the consequent surgery is technical and therefore easier".
- Principle No. 35 "The computer can show you what you achieve with your planning but cannot plan for you".
- Principle No. 36 "It is not difficult to find a solution to a problem, it is only difficult to identify the problem".
- Principle No. 37 "A necessity can neither be forbidden nor be eliminated by ignoring it".
- Principle No. 38 "In a case of micromandibulism the mandible does not need elongation only but also widening".
- Principle No. 39 "In a case of macromandibulism the jaw must not only be set back, but both halves must also be narrowed".
- Principle No. 40 "Leave as many teeth in the alveolar process only as can be positioned in their proper angulation to their respective jaw base. Alternatively, elongate the jaw base within the tooth-bearing area".

- Principle No. 41 *"Make an asymmetrical facial skeletal deformity first into a symmetrical one. Then it is either already corrected or it becomes easier to be corrected"*.
- Principle No. 42 *"Pray every day to God that you do not become blind with open eyes"*. (L. Obwegeser sen.)
- Principle No. 43 *"Learn to see, not only to look at"*.
- Principle No. 44 *"The face is an aesthetic and functional entity composed of a bony framework and soft tissues. The maxillo-facial surgeon, should therefore be able to plan and treat the face as a whole, not parts of it only"*.
- Principle No. 45 *"Plan the dysgnathic case in such a way that the desired profile can still be achieved even if the jaws were to become edentulous; otherwise problems with full dentures are preprogrammed"*.
- Principle No. 46 *"What you don't have in your brain, your hands cannot do"*.
- Principle No. 47 *"The individual human facial appearance should blend with his general skeletal type and even his character"*.
- Principle No. 48 *"For profile planning have the tracing of the cephalogram with the Frankfort plane in the horizontal position"*.
- Principle No. 49 *"In planning a case, reposition the jaws on the cephalometric tracing to fit the desired profile. That will give you the measurements of the necessary movements on the model operation"*.
- Principle No. 50 *"Maxillofacial anomalies should be corrected as early as possible and as late as necessary"*.
- Principle No. 51 *"When doing osteotomies within the dental arch, use the chance to close the gap of a missing tooth. When you have to create or leave an interdental space, then it must have the width of one or more teeth but not half of a tooth or one and a half"*.
- Principle No. 52 *"To recognize the negative is the first step in creating the positive"*.
- Principle No. 53 *"Always think why you are doing it that way and not in another way"* (H. Gillies 1957).
- Principle No. 54 *"Mandibular antealveolism is better corrected surgically than orthodontically if it is the cause of a lip ectropion"*.
- Principle No. 55 *"When performing osteotomies create broad areas for adaptation"*.
- Principle No. 56 *"Do not advise any treatment using models, photographs and X-rays and other specific tests only. The clinical assessment of the case is equally important"*.
- Principle No. 57 *"For the reconstruction of mandibular defects the iliac crest graft is the best substitute when proper function is to be achieved"*.
- Principle No. 58 *"If you want a reconstructed mandible to grow, you must achieve proper function. The same is true for cases with ankylosis"*.
- Principle No. 59 *"A graft covered by thick cortical bone is good for stability but its survival rate is poor"*.
- Principle No. 60 *"In hemifacial microsomia there is always a 3-dimensional hypoplasia of the bony and soft tissue structures of the affected region. Therefore, the correction must be accordingly"*.
- Principle No. 61 *"When your standard procedures present limitations, find another one"*.

Philosophy of Corrective Surgery Planning

It is not my intention to explain in every case why I corrected the anomaly in the way I did. Another surgeon may do the same case differently and the patient's orthodontist may also have his own ideas. The final result of the surgical correction will be evaluated by the patient himself and his friends and for them it is the surgeon's product, whether it is a good result in their eyes or not. As the surgeon is finally responsible for the outcome of the correction he should make the plan for the correction and not someone else. Based on my experience I am of the opinion that: *"Treatment planning of facial skeletal anomalies is an intellectual product based on fundamental knowledge of the subject, experience, imagination and intuition. Executing the consequent surgery is manual work and therefore easier"* (Principle No. 34).

Whenever possible it is best to discuss the case together, the orthodontist and the surgeon, before any treatment starts. In my experience it is wrong when the patient is referred for the first time to the surgeon after the presurgical orthodontic treatment is finished. Then the surgeon will be able to produce a good occlusion with osteotomies but *"A good occlusion does not automatically produce a good profile and vice versa"* (Principle No. 11).

Nowadays computer planning only is often used. When I visited one of the pioneers of computer planning he demonstrated a case which he had operated on according to his computer planning. I told him that I would have wanted another result and he agreed with my plan. That means: *"The computer can show you what you achieve with your planning but cannot plan for you"* (Principle No. 35). I am of the opinion that *"In the correction of facial skeletal anomalies the surgeon's eyes, experience and intuition are more important than tracings and computer-planning"* (Principle No. 33).

I have often seen poor results due to inadequate planning. Once a young American orthodontist, working in Germany, referred a young girl for my opinion. It was a straightforward case. I wrote him my exact treatment plan, which included the extraction of an upper premolar on each side as a maxillary advancement on each side was necessary because of her midfacial flatness due to a micro-retromaxillism, in addition to a mandibular retropositioning. One and a half years later

he accompanied the patient for surgery. He explained to me that he had learned how to plan the correction of maxillo-mandibular anomalies on a postgraduate course and, therefore, felt extracting two upper bicusps was unnecessary in this case. He had produced two well-fitting dental arches by overexpanding the maxillary arch.

He had probably not been taught that *"Overexpanding the maxillary arch cannot correct retromaxillism, nor can retruding the mandibular teeth correct ante-mandibulism"* (Principle No. 24). Instead of advancing the maxilla as I had suggested I could only camouflage the midfacial flatness by inserting L-shaped pieces of lyophilized cartilage paranasally. The aesthetic result was acceptable, but not as I had planned it. Eleven years later the young lady, now 29 years old, came back with her father as all upper front teeth were loose because of the former orthodontic overexpansion of the upper dental arch. The result will be a full upper denture in the near future. For that, the maxilla has to come forward, even if dental implants are used. It proves the principle that *"Inadequate planning may easily end up with a disastrous result, functionally as well as aesthetically"* (Principle No. 26).

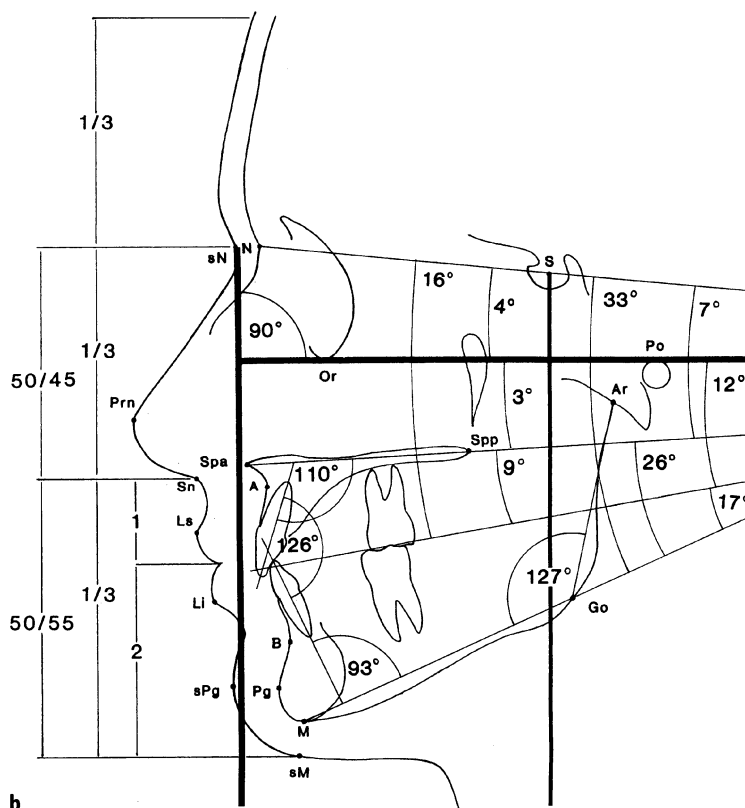
The following principle must be generally applied: *"Plan the dysgnathic case in such a way that the desired profile can be still achieved, even if the jaws were to become edentulous; otherwise problems with full dentures are preprogrammed"* (Principle No. 45).

For the actual planning one must keep in mind *"The face is an aesthetic and functional entity composed of a bony framework and soft tissues. The maxillo-facial surgeon, therefore, should be able to plan and treat the face as a whole, not just parts of it"* (Principle No. 44) and *"Patients with a facial anomaly primarily wish to have a normal appearance"* (Principle No. 1).

The planning of a case therefore starts with the evaluation of his/her face in relation to his/her general skeletal habitus. That result and also the desired profile type and facial contours are recorded. Then a sheet of transparent paper is fixed on the actual size profile as well as front view photographs of the patient. They should be made by placing a tracing of the lateral cephalogram under the enlargement apparatus and then enlarging the photographs to that size.



a



b

Fig. 10a,b. Planning the desired profile line. **a** For profile planning, the cephalometric tracing must be fixed with the Frankfort plane in the horizontal position. The new profile line differs very much and so would the plan of surgery when the tracing is fixed with the Frankfort plane obliquely (from H. Obwegeser 1981). **b** The skeletal framework and its relations for a good profile, with the desired vertical proportions

Next the surgeon himself, or even an artist, corrects the patient's profile line in accordance with the surgeon's notes. Whoever does this profile planning has to keep in mind that *"The human facial appearance should blend with his general skeletal type and even with his character"* (Principle No. 47). *"For profile planning ensure the tracing of the cephalogram has the Frankfort plane in the horizontal position"* (Principle No. 48). This is mandatory for drawing the new profile line. If the Frankfort plane deviates from the horizontal, wrong profile planning is unavoidable causing wrong amounts and directions of movements of the mandible and the maxilla, as clearly seen in Fig. 10a. *"In the treatment planning of facial anomalies, first the profile, then the occlusion"* (Principle No. 8).

When drawing the new profile line one has also to keep in mind the artist's rule of the relation of the three facial thirds (Fig. 10b). For planning the amount of the necessary movement of the jaws one has to consider how much the soft tissues follow the jaws and the teeth when they are moved.

An additional tracing of the maxilla, mandible and teeth is used to cut out the jaws separately and move them into such a position that the planned profile will result, independent of the dental situation, following the principles: *"In planning a case, reposition the jaws on the cephalometric tracing to fit the desired profile. That will give the measurements of the movements necessary in the model operation"* (Principle No. 49) and *"If alterations of the bases of the facial thirds are required, dental alignment and the occlusion must not limit the goal set"*

(Principle No. 8). That means *"When planning the correction of a maxillo-mandibular anomaly, consider the jaws as if they were edentulous. Then you will be able to bring their bases into proper relationship and not the teeth only"* (Principle No. 10).

With the help of computerized technology the planning can even be more precise as long as we tell the computer what we want. But we have to decide which type of profile is desirable. Then the computer may be able to tell us what parts of the facial skeleton have to be moved, in which direction and by how many millimetres.

The amount of movement of the maxilla and the mandible is transferred to the model-operation. For that, a pair of models are fixed in an articulator in accordance with the registration of the mandibular movements in the joints. This model-operation will give the answer to the question whether or not, with the amount of movement of the jaws gained with the tracing planning, the desired occlusion can be achieved. If not, that model-operation will indicate how many teeth have to be extracted in order to place them in proper angulation to the respective base of the jaw and produce a normal curve of Spee, as a prerequisite for achieving a good occlusion with the desired amount of movement of the jaws. With that final planning, the orthodontist will know what he has to achieve, and the surgeon will know what kind of corrective surgery he will have to perform. After that, both specialists are able to inform the patient in detail.

For details of practical planning see case of Fig. 63.

Mandibular Asymmetries

The symmetrical form and positional anomalies of the mandible and its principal parts do not produce great difficulties in diagnosis or in therapy because of today's diagnostic and therapeutic possibilities. However, as soon as the form of the mandible develops asymmetrically the diagnosis can become rather difficult.

The asymmetries of the mandible are a special group of jaw anomalies. They develop either embryonically or postnatally, during the general growth period or occasionally even later.

Three groups can be distinguished.

The *embryonic forms* are either due to condylar aplasia or hypoplasia or they appear as evidence of a spontaneously occurring (hemifacial microsomia) or a hereditary anlage-induced disturbance of development as part of a syndrome (Treacher Collins, etc.). We were never able to identify with certainty an anlage-induced or genetically proved aetiology of a mandibular asymmetry only.

The *postnatally-induced disturbance of development and growth* can be the consequence of an adverse event during growth affecting one hemimandible only, or both unequally. In other cases the abnormal growth seems to be the consequence of a *misregulation of growth without any recognizable cause*.

These three groups of mandibular asymmetries appear again and again in the same forms typical for each group, however, they are variable in extent depending on the aetiology.

In the first group, the embryonic maldevelopment of the mandible, we have to distinguish between two subgroups. In those congenital forms which are due to an aplasia or hypoplasia of the condyle an otherwise almost normal mandible can develop. In these cases the asymmetry is mainly due to lack of height of the ascending ramus as a consequence of the absent condyle or a part of it which reduces the height of the ascending ramus by that amount. It does not have any really negative influence on the development of the main part of that hemimandible. This fact seems commonly known (D.H. Enlow 1982). The lack of ramus height leads, as a consequence, to the development of an oblique occlusal plane. In the cases of condylar hypo- or aplasia within a syndrome, the whole hemimandible seems to be influenced negatively in its development. In their appearance they are very similar,

but they are diagnosable as a more or less typical form of the syndrome of which they are a part.

The second group comprises all those mandibular asymmetry forms which develop postnatally following damage to the condyle. The forms of the mandibles of that group are also very similar to each other, depending mainly on the degree of damage and the developmental stage of the mandible, and appear almost independent of the type of the adverse event. All cases of this second group produce mandibular asymmetries due to hypoplasia of the abnormal side which is called hemimandibular hypoplasia.

The third group of mandibular asymmetries is characterised by surplus growth in length, mass or both of the affected hemimandible. This third group is undoubtedly the most interesting one. It contains the largest variation in graduation either as surplus in length, in mass or in both of the affected hemimandible. This third group of mandibular anomalies is caused by a postnatal condylar misregulation of growth. It was first extensively described and classified by H. Obwegeser and M. Makek in 1986. When, additionally in such cases, an asymmetry is also produced on the contralateral side by the same aetiology, then the picture can become very complex.

In the following the most important aetiological possibilities are classified.

8.1 Aetiology of Mandibular Asymmetries

- Embryonic genetic
- Embryonic non-genetic
- Postnatal adverse events during growth
- Adverse events after growth has ceased
- Misregulation of growth after birth

8.1.1 Mandibular Asymmetries Due to Embryonic Growth Abnormality

- Unilateral condylar hypo- and aplasia
- Unilateral mandibular hypo- and aplasia
- Unilateral mandibular and facial hypoplasia
- Hemifacial microsomia
- Hemifacial hyperplasia

8.1.2**Mandibular Asymmetries Due to Adverse Postnatal Events During the Growth Period**

- Trauma
- Arthritis
- Osteomyelitis
- Ankylosis
- Irradiation
- Perimandibular haemangioma
- Tumour or tumour-like lesions

8.1.3**Mandibular Asymmetries Due to Misregulation of Growth After Birth**

These may be due either to hyper- or hypoactivity of the local condylar growth regulation mechanism on one or both sides of the mandible. There are several possibilities:

- Hemimandibular hypoplasia
- Hemimandibular hyperplasia (H.H.)
- Hemimandibular elongation (H.E.)
- Hybrid type of hemimandibular elongation and hemimandibular hyperplasia
- Unilateral
- Bilateral
- Combinations

8.1.4**Mandibular Asymmetries Developing After Growth Has Ceased**

- Hyperactivity of condylar growth factors
- Acromegaly
- Trauma
- Chronic osteomyelitis, primary and secondary
- Simple hyperplasia of the condyle, or hyperplasia of arthrotic or tumorous origin
- Tumours and tumour-like lesions of the mandible

What Do We Know About Growth of the Mandible

Does our present-day knowledge concerning growth and development of the mandible permit us to explain easily how these asymmetrical anomalies of the mandible develop? It is not intended to repeat and discuss here the complex subject of the growth of the various bones of the complete facial skeleton. For that we may use the various well known publications on the subject of facial growth (D.H. Enlow 1982, 1990; A. Björk 1955; A. Björk and V. Skieller 1977; Björk and Skieller 1983; A. Brodie 1941; M.L. Moss 1962, 1968; M.L. Moss and P. Salentijn 1969; D. Precious and J. Delaire 1987; P. Stöckli 1994). On the other hand, extensive clinical experience with patients relating to the growth of the mandible may bring additional knowledge to the complex problem of mandibular growth and in particular the role of the condyle. Of course, the mandible cannot be viewed, from the growth physiology point of view, as an isolated piece of bone independent of its surroundings and their influence, although one might often form the impression that this is so.

The bones of the facial skeleton grow by apposition and resorption. Out of that process develops the shape of each bone. The definitive form of the facial bones is not defined at birth. It changes during the growth period influenced by the genetic inheritance, the surrounding soft tissues and possible pathological conditions. The process of apposition and resorption is guided by the sutures except in the mandible. The cranium and the orbits are influenced in their growth, development and shape in addition by the increase in their contained soft organs such as the brain and the orbital contents as well as by the covering soft tissues. This fact is particularly obvious in neurological diseases of the facial musculature and also in cases of hypo- and hypertrophy of the attached musculature.

According to M.L. Moss (1962), it is the “functional matrix” which allows the facial bones to enlarge. This does not explain the simultaneous development of “micromaxillism” and “macro- or antemandibulism” in the same patient. In my opinion these anomalies are only understandable on the basis of a genetic steering mechanism. However, according to clinical experience we do not doubt that the functional matrix, comprised of the musculature attached to a certain part of the facial skeleton, may influence its growth and development in

shape very remarkably. This is true in the positive as well as in the negative sense.

Postnatally, the growth of the viscerocranium develops mainly parallel to the general growth. The growth curve of the facial bones is rather steep during the first 4 years of life, afterwards becoming somewhat flatter, but steeper again during puberty. The detailed growth spurt differs between boys and girls in quantity as well as in age. It is at its peak at the age of 14 in boys and its highest intensity is at the age of 12 in girls. In addition, the absolute increase in growth is remarkably greater in boys (M. Riolo et al. 1974).

The wrist radiograph permits determination of the age of development, the so-called skeletal age and that allows prediction of the amount of growth to be expected, as already mentioned when discussing the hand-wrist radiograph in the documentation chapter. Although the growth curve of the facial bones basically parallels that of body height, one has to consider that the end of growth of the facial bones stops about 6–12 months later. The neurocranium has already reached 85% of its final size by the age of 5 years. The bones of the middle third of the face finish growing at about the age of 17 years, i.e. about a year earlier than the mandible. In males the mandibular growth can continue up to 23 years. Under pathological growth influences it can start again. Examples are acromegaly and H.H. and H.E. and their hybrid forms.

9.1 Body and Ascending Ramus of the Mandible

The mandible consists of two halves. It has a dual developmental start. The starting nucleus of the mandible is Meckel's cartilage. The ossification of the mandible begins from embryonic connective tissues around Meckel's cartilage. It does not replace that cartilage but builds on it. The main part of Meckel's cartilage does not ossify, it remains in a groove medial to the mylohyoid crest until it regresses in the 23rd embryonic week. Its perichondrium becomes the sphenomandibular ligament. The duplicated mandibular bones unite at the symphysis. This ossifies in the second year of life.

At birth there is no real gonial angle. That only develops, like the articular prominence and the glenoid fossa,

when the teeth erupt. This latter statement is true only when the ramus and condyle develop normally.

During the prenatal phase the mandible stays in a pronounced retroposition. This is self-correcting during the course of postnatal growth. The condylar cartilage of the mandible shows an enchondral type of growth. However, it cannot readily be put on a par with an epiphyseal suture of the long bones. It has various structural and histochemical differences and reacts differently under experimental conditions. It is definitely not responsible alone for the growth of the mandible. There is additional periosteal and endosteal growth activity along the whole mandible.

The growth of the mandible is also a result of apposition and resorption and remodelling. In its growth physiology it is only partially comparable with that of the other facial bones or the long bones. One has to look at the growth of the body and ascending ramus separately as these two parts of the mandible do not develop in the same direction although the ascending ramus seems to “grow out” of the mandibular body. The ascending ramus grows upwards and posteriorly thus pushing the mandible downwards and forwards. The body elongates in an antero-posterior direction by periosteal and endosteal apposition of bone in both directions.

9.1.1 The Angle of the Mandible

Since the mandibular body and ascending ramus grow in different directions, it is their growth which is responsible for the formation of the mandibular angle. However, since the body grows mainly horizontally, it is the direction of the ascending ramus which is responsible for the shape of the angle of the jaw. On the other hand, the direction of the ascending ramus depends on the growth direction of the condyle. And what determines its growth direction? Can it be influenced during mandibular growth or even after? It is believed that the position of the middle cranial fossa determines the position of the condyle which in turn influences the direction of the condylar neck as well as the direction of the ascending ramus. I have my doubts about this because, as a consequence, all cases with basal craniostenosis, in particular the Morbus Crouzon and Morbus Apert cases, would always produce a similar form of the mandible. I have not observed this. More plausible seems the explanation that the form of the gonial angle, be it flat or otherwise, depends upon whether one is a “horizontal grower” or a “vertical grower”. When the patient is a “vertical grower” in general height, this means that the hypothesis cannot be supported. Therefore, it only remains to confess that we do not know what is responsible for the great variation in size of the angle between mandibular body and ascending ramus.

Certain osteotomy techniques permit us to alter the form of the gonial angle. Sometimes the desired result is stable and sometimes the angle becomes flat again. There are still many things we do not yet know. It is my belief too that the musculature attached to the mandible plays the dominant role in forming the mandible and its angle always within the genetic pattern. Abnormal muscular situations, congenital or acquired, and in particular surgically altered ones are capable of altering the already fully developed form of a mandible especially of its angle. Unfortunately most of these changes are in a negative manner. And yet there is proof that in the condyle a growth regulating system must exist which can influence the size of the gonial angle. The proof is the fact that in cases of H.E. slender type, we expect to find a somewhat or very stretched gonial angle while the contralateral side may have a gonial angle within the normal range. H.H. produces an almost rectangular gonial angle. However, it will be rounded off.

9.1.2 The Chin

The development of the chin has to be regarded as almost independent of the mandibular body. In infancy and early childhood it is relatively smaller and more retruded than the body of the mandible which itself is always in a retruded position and only grows forward during further growth. Independently of the amount of anterior development of the mandible, the chin can develop into an ante- or normo- or retrogenia in spite of the fact that the mandible ends up in “normomandibulism” or “antemandibulism” or even “retromandibulism”.

What do we know about how the chin develops? During the growing period of the facial skeleton “...bone is progressively added onto the external surface of the basal bone area including the mental protuberance” (D.H. Enlow 1982; Chap. 3, Part 2). How can it be explained that in one person the quantity of that bone apposition is so much larger than in another? We know through clinical experience that in all cases of severe damage to the condyle in the early growth period limiting proper function a severe retrogenia is always found. This retrogenia is independent of whether the cause has been inflammation, ankylosis after trauma, non-reduced bilateral luxation of the condyles or a so-called condylolysis of whatever origin (see cases Fig. 13, Fig. 23, Fig 24, etc.). But we can also find severe retrogenia or almost lack of a mental protuberance in the case of anlage-induced micromandibulism in spite of the existence of normal condyles. And we can find normogenia in a case of congenital lack of a condyle (case Fig. 15) as well as after removal of a traumatically destroyed condyle in early childhood resulting in perfect mandibular function (case Fig. 26). So what do we really know about the growth and development of the chin? Not much!

9.1.3

The Alveolar Process

It is generally accepted that the alveolus develops as a result of the teeth erupting from within the mandibular body. Therefore, the alveolus becomes wider in the molar region than in the premolar or even in the incisor regions. In spite of this, there may still be a variation in alveolar bone, both in height and width, from one patient to another. It is also said that the shape of the alveolus is very much influenced by the surrounding soft tissues, the musculature, soft tissues of the cheeks, lips and the tongue. There is no question about this in cases with muscle pathology such as muscle spasm. Also in "normal" cases there may be a difference in the tension of the facial soft tissues. Whether these factors also influence the size, volume and position of the alveolar process, I am unable to say. To me it seems rather a matter for speculation.

In cases of ante- or retroalveolism it may be the position of the erupting teeth which produces the various types of alveolar positional abnormality. The tongue can have a very severe negative influence on the position of the alveolar process with its contained teeth. It can push the alveolus including the teeth forwards and laterally. True macroglossia has more power to do this than does pseudo macroglossia (P. Egyedi and H. Obwegeser 1964). The tongue itself can never be the cause of skeletal relapse after a setback operation for the correction of antemandibulism. In the late 1950s we reduced the tongue of every antemandibulism case prophylactically before the set back operation. We learned that this was totally unnecessary. Even in cases of true macroglossia the relapse will not be a skeletal relapse but an occlusal relapse because it is only the alveolus with the contained teeth which is tilted by the pressure of the tongue. Even screws and plates cannot prevent the relapse of a corrected antealveolism in cases with macroglossia unless the tongue is primarily reduced in size.

The amount of vertical growth of the alveolar process can differ quite remarkably from one patient to another. Again the cause for this difference is not known. However, in cases with a deep overbite, we can speculate that the very reduced vertical growth of the alveolar process may be due to the strong chewing musculature since in these cases we always have a gonial angle of almost 90°. The consequence of this would be that in all cases of true masseter hypertrophy we should also find lack of vertical alveolar growth. But this is not always the case. So again there are many things in the growth of the alveolus for which we do not have a clearly acceptable explanation.

The alveolar process cannot be clearly demarcated from the mandibular base, not in the fully developed state with all its teeth, nor after the loss of the teeth when the alveolar bone undergoes involution. We all

know that there are patients who retain a well-formed alveolar process for the rest of their lives after loss of the teeth and others who lose the alveolar process completely so that only a very atrophic mandibular body is left with the mandibular nerve lying on the surface and leaving the base of the mandible only. Why that nerve is wrongly termed the "inferior alveolar nerve" I do not know. It is never situated at anytime of life within the alveolar process but always within the body of the mandible. For that reason it should be termed the "mandibular nerve". This was agreed by anatomists with whom I have discussed this matter.

9.1.4

The Muscle (Coronoid) Process

The shape and length of the muscle process may be the result of inherited influence on one side and the musculature on the other side. It can be short and rounded, long and pointed or anything in between. Why it occasionally elongates so much that the mouth opening is remarkably reduced because of its inability to pass beneath the zygoma and its arch, we do not know. Patients can almost never clearly define the onset of the reduced mouth opening. Their usual answer to the question of their age when they first noticed it, is, in general: "It was always like that". If it were to develop during growth one might imagine that the pull of the temporalis muscle would be strong enough to cause the coronoid process to develop in such a direction that the mouth opening would not be hindered. That permits the conclusion that the abnormal elongation of the coronoid process is of embryonic developmental origin.

It is not known as a sign of a syndrome and it is also not known to be of clearly inherited origin.

Its symptomatology and its surgical correction have already been mentioned.

9.1.5

The Condyle and Its Function

Much has been published on the function of the condylar head and particularly its cartilaginous layer. There are two view points to be considered when discussing the condyle and its function. One is the anatomical function of it and the other is its possible influence on the growth and development of the mandible (J. Delaire 1990). Its main anatomical function is to support the mandibular body during mastication and to permit proper mobilization of the mandible.

The condylar head is very much like the head of a bone of the extremities, however, with the articular disc interpositioned between the head and the fossa. The head with the disc is responsible for movements in the joint. Its cartilaginous layer serves like that of other joints by absorption and diminution of the mechanical

forces affecting the joint during function. Unquestionably, the cartilage layer also has the potential and purpose of producing regional adaptive growth of this bone. With that capability the often-observed change in shape of the condyle after repositioning osteotomies can be explained.

It seems just as certain that the cartilage layer also possesses an active function in growth regulation. Only by this fact can it be explained that histologically as well as by radioisotope studies, massive cell hyperactivity can be identified within the cartilage layer during the active phases of growth. That provable activity disappears after termination of growth. During the active phase of the development of a H.H. or H.E. or their hybrid forms, pronounced hyperactivity of that cartilage layer can be observed by scintigraphy with Technetium 99 DPD. During the growth spurts of the mandible, an evident activity of the condylar cartilage zone can always be proven and this is particularly striking during the development of one of the misregulations of the condylar growth steering mechanisms.

There is also the fact that the changes evident in the shape of the mandible in H.H. and H.E. happen to one half of it exclusively and end exactly at the symphysis. This suggests that these changes in shape of the mandible must have a local origin within that mandibular half. That this cause must be located within the superficial region of the condyle is, as already mentioned, proven by the fact that very active changes in shape of a mandibular half as seen in H.H., H.E. or their hybrid forms, immediately comes to a standstill following high partial condylectomy.

If an intracapsular high condylectomy only is performed, postoperative function should not cause any major problem. In spite of high condylectomy, further growth and development of that mandibular half proceeds undisturbed as long as normal function exists.

Because of the existence of H.H. and H.E. we are convinced that the condylar cartilage layer must contain at least two growth regulators which produce different effects. One has the capability of stimulating growth in length and the other of production of bone mass. We call that factor or stimulus or regulator which is responsible for length of growth, regulator L and the one responsible for the production of bone mass as regulator M. In cases with undisturbed growth of the mandible, both condylar growth regulators seem to be either inactive or in balance in their regulatory activity. As a consequence, only the genetically determined growth occurs.

Why and how that balance can be disturbed and the regulators go into hyperfunction, we do not know.

The condylar cartilage zone is definitely not a growth centre entirely responsible for mandibular growth, although we believe that it contains regulators which can give rise to abnormal growth. The fact that after condylectomy for whatever reason, the half of the mandible operated on can develop in growth equal to the non-operated side speaks against the existence of a "single growth motor" within the condyle. The normal growth of the mandible does not come to a standstill after high condylectomy. The contrary can be the case. The removal of a traumatically damaged condyle permits the restrained growth of the mandible which is due to damage to the condyle, to grow normally again. What growth has already been lost will indeed not be made up but there will be no additional growth deficit. We have observed this again and again, when in childhood, in cases of partial or total ankylosis of the TMJ the traumatically damaged condyle was removed in order to achieve normal mouth opening and restore normal mandibular function. But we have also observed further lack of growth after successful surgical release of a complete posttraumatic TMJ ankylosis, however, without the removal of the destroyed condyle. Consequently a condyle traumatized or damaged by infection or inflammation can have a negative influence on the growth of the mandible on the relevant side by slowing the growth.

However, normal growth of the hemimandible is also possible in cases of unilateral congenital lack of the condyle. Our case in Fig. 15 seems to verify this and other authors also demonstrate this (D.H. Enlow 1982; Chap. 3, Part 2).

Fracture of the condylar neck with dislocation of the condyle and also intracapsular fragmentation of the condyle do not automatically lead to impairment of growth of the mandible. In cases when normal function results as a consequence of the treatment, usually no disturbance of growth of the mandible is observed. Therefore, without any doubt, function is a very important growth stimulus. Further supporting evidence is that after resection of the ramus including the condyle and simultaneous reconstruction with an iliac crest graft, in early childhood, that piece of grafted bone increases in length equal to the non-operated side (see case in Fig. 27). However, an iliac crest graft does not possess a growth centre or a growth regulation mechanism or an epiphysis. Consequently only function can be responsible for the growth of such a graft.

Clinical Experience Regarding the Influence of the Condyle on the Growth of the Mandible

The mandible is normally symmetrical in size and shape, but in clinical practice cases are often seen which differ from the norm. These deviations from normal stimulate us to enquire about their aetiology and their growth mode, both in respect of the symmetrical cases as well as the asymmetrical ones. In particular the asymmetric forms are the ones which are of special interest both in their aetiology and in form development. They can be forms with a growth deficit as well as those with an excess of growth. As far as the plus or minus of growth of a complete mandibular half is concerned, the cause must lie in the growth regulation mechanism within that mandibular half. This means there must be a local factor independent of whether both or one side only is involved. This is true for those growth anomalies with a known cause as well as those of uncertain aetiology. As long as one agrees that the condyle with its cartilaginous zone can have an influence on regulation of growth of the mandible, and of that there is no doubt (see Fig. 28), it seems possible that condylar hypoactivity as well as hyperactivity can exist. Therefore, both have to be differentiated from normal activity which is responsible for the normal growth of the mandible within the genetic pattern.

Normal activity as well as hypo- and hyperactivity are terms relating to function. How can the variations in function be proved? Hyperactivity can be clearly demonstrated with radio-isotopes and/or histologically. The

same is also true for hypoactivity, however, only when it is possible to compare the findings with the normal ones. In addition to scintigraphy and histology the size and shape of the condyle may help in making the diagnosis. When a hypoplastic condyle is found, the clinician is accustomed to expecting hypoactivity with reduction of growth of that mandibular half leading to hemimandibular hypoplasia.

How can we define or ascertain normal condylar activity? Condylar activity can at present only be ascertained histologically or by scintigraphy. To my knowledge, serial investigations showing the histological picture of the condylar cartilage zone taken in the important phases of growth of the mandible do not exist in large enough numbers for the various age groups, to allow valid comparisons.

It is possible, with the help of scintigraphy, to obtain information on the amount of condylar cartilage activity in cases of undisturbed mandibular growth in the various age groups. That information permits comparison of the normal average findings with those of the patient in question. This is of great benefit both in cases with uni- and bilateral hypoplasia as well as in the condylar hyperactivity cases. We owe this knowledge to Kaban et al. (1982) who have gone into that subject as mentioned in the chapter on "Scintigraphy, a Diagnostic and Treatment Planning aid" (see Chap. 5). It has not yet become a routine procedure.

10.1 Condylar Hypoactivity

When hyperactivity of two different growth regulators exists within the condylar cartilaginous zone, and there is no doubt about that in my opinion, then hypoactivity of these growth regulators should also exist. That hypoactivity should then either affect that regulator which is responsible for H.H. or on the other hand the one which produces H.E. Therefore, two different types of hypoplasia of one half of the mandible should theoretically be observable.

No doubt we always find hemimandibular hypoplasia in connection with condylar hypoplasia with the exception of hereditary congenital hemimandibular hy-

poplasia (see case Fig. 11). From that we conclude that condylar hypoplasia results in growth hypoactivity. However, it seems that during the development of condylar hypoplasia, both growth regulators are impaired independently of what caused the condylar hypoplasia. Therefore, a hybrid form of hemimandibular hypoplasia should result. This seems likely as there exists no clear hemimandibular hypoplasia specific to one of the two growth regulators.

When damage to the condylar head occurs during the phase of development and growth of the mandible, subsequent lack of growth and a more or less typical

form variation of the involved mandibular half may result, generally called hemimandibular hypoplasia. This is true, whether or not the damage is embryonic or postnatal, and is independent of either origin. It seems, therefore, that independent of the cause of damage to the condyle in infancy or childhood, a similar picture of disturbance of mandibular development of the affected half of the mandible always results. Obviously the various degrees of damage are always mediated through the same steering mechanism, since a very similar form of growth and shape of the mandible always results. Only the amount of disturbance of growth varies which is clearly dependent on the age and to what degree the condyle was damaged. Due to the damage to the condyle, condylar hypoplasia results. As we believe in the proven active role of the condyle on the growth and development of the hemimandible, it seems that its activity is reduced proportional to the amount of damage.

Naturally, this damage to the condyle with consequent lack of growth of that hemimandible as a result of its hypoactivity is much more obvious when it exists unilaterally rather than bilaterally. A very clear exception in the appearance of the form of the mandibular side involved is seen in isolated embryonic hypo- and aplasia of the condyle when they are not part of a syndrome.

10.1.1

Bilateral Condylar Hypoactivity

Bilateral condylar hypoactivity can either have an embryonic origin or it is acquired postnatally. It results in hypoplasia of both mandibular halves. The alteration in shape of each mandibular half is equal to that of the unilateral form, again depending on the amount of intrauterine or acquired condylar hypoactivity. In the non-hereditary cases, the degree of damage to the condyle and the time of onset during the embryonic or postnatal phase of development and growth is decisive for the amount of condylar hypoactivity and consequently for the amount and form of the hemimandibular hypoplasia. In these cases, the mandible will be too small in all dimensions, the condyles are hypoplastic and the prominence of the chin is too short in depth and retruded. The axis of the lower front teeth is tilted and the teeth are crowded as they do not have enough space in the much too short mandibular halves. The patient presents the so-called birdface appearance. The genetically determined form and those due to symmetrical disturbance of development as part of a syndrome (Treacher Collins) produce a different, easily distinguishable bilateral hypoplasia. This is also the finding in patients with bilateral unrepositioned luxation of both condyles and the so-called bilateral condylolysis (G. Rabey 1977, 1978) both when occurring in early infancy and also in the event of bilateral ankylosis in the growth period. In cases with asymmetrical damage to the con-

dyles, in cases with bilateral hemifacial microsomia, after radiotherapy in early childhood and also after bilateral juvenile arthritis (Morbus Still), a bilateral asymmetric micromandibulism can develop.

In cases with symmetrical bilateral hypoplasia, naturally no oblique occlusal plane will develop. However, since both rami grow too short on each side, the maxilla cannot develop downwards on both sides in the posterior region. As a consequence a bilateral more or less steeply-ascending occlusal plane will develop.

Four cases of bilateral condylar hypoactivity each with a different aetiology will demonstrate the problems.

Congenital Bilateral Condylar Hypoactivity

There are cases with congenital bilateral hemimandibular hypoplasia, both symmetrical as well as asymmetrical. They are obviously due to intrauterine trauma to the foetus.

This is the case in the hereditary Francescetti Syndrome. It is a symmetrical type of bilateral hemimandibular hypoplasia in full definition of the word hypoplasia. Whether this is secondary, due to damage to the condyles, or as part of the whole skeletal and soft tissue abnormality is another question. Since the zygoma, the eyelids and ears and even the palate are involved, the mandibular hypoplasia might be a part of the whole abnormality rather than due to intrauterine damage to the condyles only. The Francescetti Syndrome type of bilateral hemimandibular hypoplasia is often seen. Therefore, there seems little reason to illustrate such a case.

In the case of bilateral hemifacial microsomia, the mandibular hypoplasia will be asymmetrical, depending on the amount of damage to the embryonic tissues on either side by bleeding from the stapedia arteries (D. Poswillo 1973) on the left and right sides. So, in these cases we should not attribute the hypoplasia of the mandible to condylar damage alone.

Anlage-Induced Bilateral Hemimandibular Hypoplasia

There are cases of bilateral, very symmetrical hemimandibular shortness, which we cannot classify as being the result of condylar hypoactivity. As far as we know, cases with condylar hypoactivity always manifest remarkably smaller than average condyles. In these anlage-induced, sometimes even hereditary, bilateral hemimandibular hypoplasia cases, we find a different size and shape of the condyles than that found in the hypoactivity cases. The condyles are rather large, but definitely within the normal size and shape. The condylar neck is shorter than normal but rather thick, in contrast to the small condyles and thin condylar necks of the hypoactivity cases. The same difference is true for the ascending ramus and body of the mandible. They are short but rath-

er thick and voluminous while those of the hypoactivity cases are thin and gracile. Although the chin is also retracted, as in the hypoactivity cases, it is high (deep), voluminous, not petite and rather short in height as in the bilateral acquired hypoplasia cases.

Because of all these anatomical facts, we have to distinguish this type of symmetrical hemimandibular hypoplasia (shortness) from the bilateral condylar hypoactivity cases.

As we do not know of any histological or radio-isotopic findings in condyles with this type of mandibular

abnormality, we are unable to say whether or not they are also due to condylar hypoactivity. In our opinion this mandibular abnormality has to be accepted as an isolated condition similar to true antemandibulism (mandibular prognathism) and true retromandibulism (mandibular retrusion).

As this type of mandibular abnormality is not an often observed phenomenon, the presentation of a typical case will be worthwhile.

Anlage-Induced Bilateral Hemimandibular Hypoplasia (Fig. 11)

Sex and Age. Male, 18 years of age.

Aetiology and History. Unknown. During the growth period the mandible did not develop to normal size. At the age of 16, the patient started orthodontic treatment in preoperative preparation for surgery. There was no relevant family history.

Clinical Findings. In all aspects, a typical symmetrical micromandibulism and hypo- and retrogenia (Figs. 11a,b). The occlusal picture was that of a class II case with large interdental spaces in the mandible (Fig. 11c), obviously after previous extraction of the first molar on each side.

Radiographic findings. Panorama view, TMJ tomograms, lateral cephalometric view (Figs. 11d–g). They

revealed a mandible short in all parts, the ascending ramus as well as the body and the chin. However, the latter was severely retruded. No special trabecular structure; the condyles were within the range of normal size and structure. The condylar necks seemed to be shorter than normal.

Diagnosis. Anlage-induced “micromandibulism” and “retro- and hypogenia”.

Treatment Plan. Presurgical orthodontics following the author's principle: “*The goal of presurgical orthodontics must be to: a) arrange the teeth in proper angulation to the jaw base, independent of the number of extractions necessary, b) do not extract teeth when there is enough*

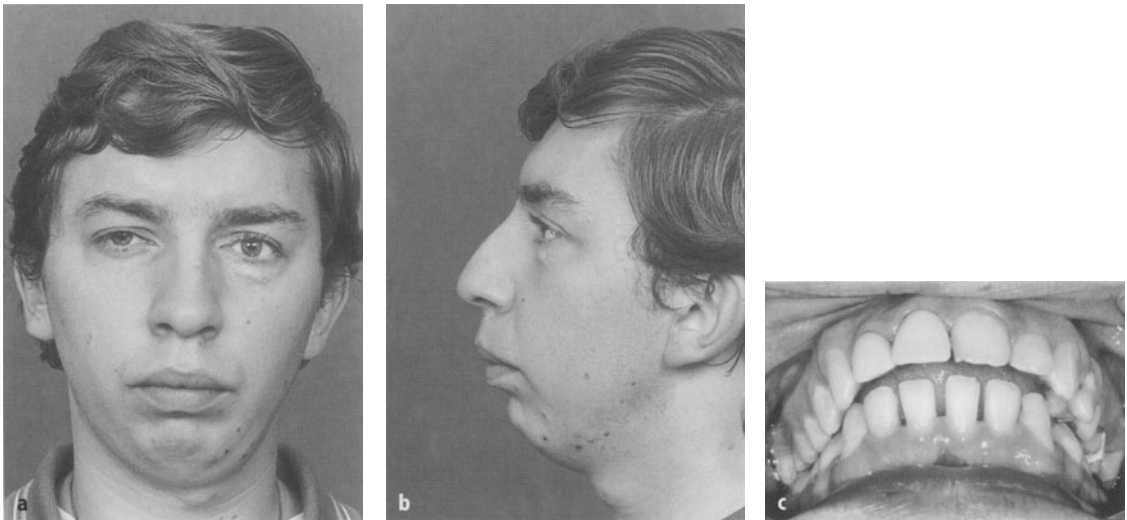


Fig. 11a–c. Case of anlage-induced bilateral hemimandibular hypoplasia with normal condyles, producing symmetrical micromandibulism with severe retro- and hypogenia. Preoperative **a** front, **b** profile and **c** occlusal view

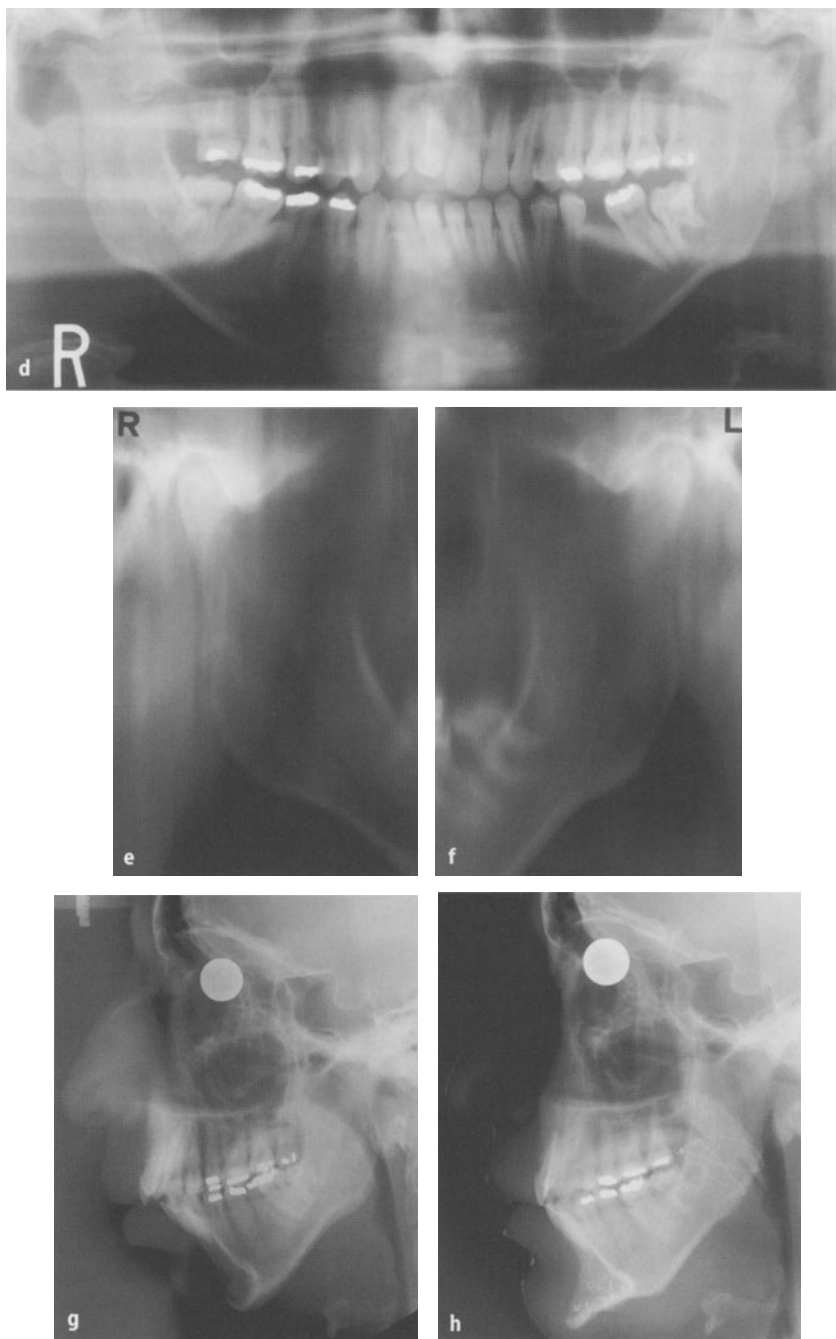


Fig. 11d–h. Case of anlage-induced bilateral hemimandibular hypoplasia with normal condyles, producing symmetrical micromandibulism with severe retro- and hypogonia. **d** Preoperative panoramic radiograph, **e** right and **f** left TMJ tomograms including the rami do not show any real abnormality of the condyles. **g** The lateral cephalogram shows the skeletal background to the patient's appearance. **h** The lateral cephalogram taken 1 year after surgery shows that normal skeletal contouring has been achieved. Note the mass of bone apposition above the two slices of chin advancement

length of the base of the jaw to place all of them into correct angulation, c) create a normal curve of Spee, d) create dental arches which will fit together" (Principle Nr. 25).

If there is enough space to bring the teeth into proper position and angulation to the respective base of the jaw, then no extractions will be necessary as in this case. After presurgical orthodontics, the author's standard procedure for birdface correction without any tooth extractions will be applied. Elongation of the mandibular body through a bilateral sagittal long-surface splitting procedure and elongation of the chin prominence by a double step, muscle pedicled chin advancement (H. Obwegeser 1988b) (Fig. 11i). Additional repositioning of the maxilla was not found necessary in this case.

Treatment Performed. As it was a standard deformity case, the treatment was performed according to the standard treatment plan. The presurgical orthodontics was kindly done by my very good friend and colleague, Dr. Paul Stöckli, after we had discussed and agreed with the treatment plan. The surgical part did not present any difficulty. The surgical procedure produced the desired elongation of the horizontal and ascending rami of the mandible including the chin and a very good occlusion was achieved as a result of the excellent presurgical orthodontics (Figs. 11h j–l).

Discussion. Micromandibulism often develops without known intrauterine or postnatal adverse influences. We must therefore label it "of anlage-induced origin". All

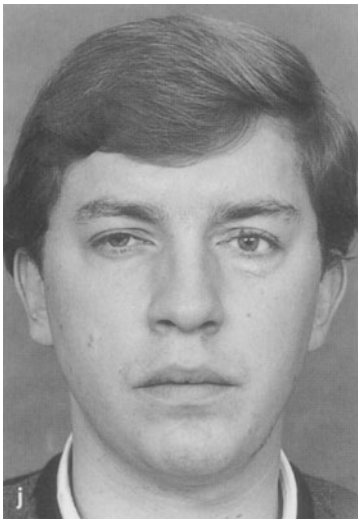
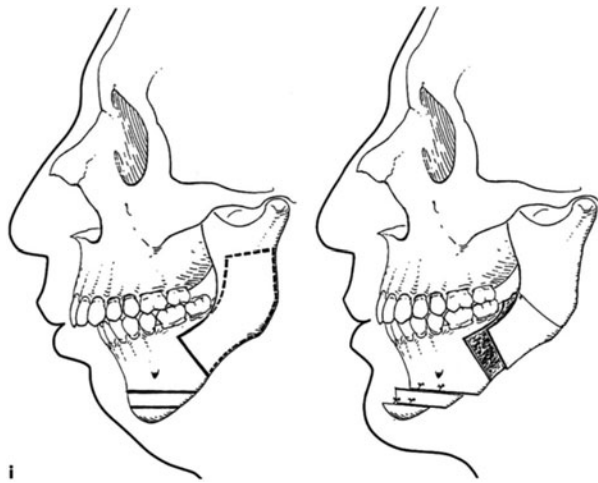


Fig. 11i–l. Case of anlage-induced bilateral hemimandibular hypoplasia with normal condyles, producing symmetrical micromandibulism with severe retro- and hypogonia. **i** Drawing demonstrating the author's one stage correction technique of such cases (H. Obwegeser 1973). Patient's **j** front, **k** profile and **l** occlusal situation 1 year after correction

these cases seem to develop a mandible of almost normal configuration but too short, with severe retrogenia and almost normal shape and trabecular structure of the condyles. It is striking that these cases produce just as short a mandible and chin as those originating from postnatal damage to the condyles. But the mandible in these cases seems less narrow and delicate than those of other origins. As the condyles in these cases show no signs of previous trauma, arthritis or irradiation injury and have a size and form within the normal range and structure, we believe that they are not due to bilateral hypoactivity of the condylar growth mechanism. It seems to be a type of anlage-induced or hereditarily determined mandibular shape, as observed in the classical cases of antemandibulism (mandibular prognathism) or the often observed hereditary retromandibulism (class II – malocclusion or mandibular deficiency case) with a normal shaped mandible and chin but overall too short as if it had not grown forward enough out of the angle area.

Conclusion. There is a type of micromandibulism with retrogenia which does not seem to have congenital or acquired hypoplasia of the condyles with a related con-

dylar hypoactivity as its cause. This type of lack of mandibular growth seems to be anlage-induced. The shapes of the condyles as well as the mandible and chin differ from those of other causes.

Acquired Bilateral Condylar Hypoactivity

Any postnatal adverse influences damaging the condylar head in infancy or early childhood may cause condylar hypoactivity. These adverse influences may be equal in their damage to the condyles thus producing a symmetrical disturbance of mandibular growth resulting in symmetrical hemimandibular hypoplasia. This is true for bilateral ankylosis in infancy whatever the aetiology might be. It may be caused by bilateral condylar fractures or infection, etc. Also, bilateral untreated luxation of the condyles in infancy may lead to symmetrical hemimandibular hypoplasia.

However, whenever the damage to the condyles is unequal, the amount of hypoactivity may differ from left to right and so may the amount of growth development on either side thus producing an asymmetrical bilateral hemimandibular hypoplasia.

Bilateral Asymmetrical Condylar Hypoactivity due to Juvenile Arthritis (Fig. 12)

Sex and Age. Female, 17 years of age.

Aetiology and History. No similar appearance in the family tree. As a child the patient suffered from chickenpox, mumps, measles and at the age of 9 years from infectious mononucleosis. Because of TMJ pain at the age of 14, the articular disc of the left TMJ was removed. At the age of 15, a silastic implant was placed in front of the chin in order to improve its prominence. The girl was then referred from overseas for correction of her retrodisplaced mandible with an open bite.

Clinical Findings. The outward appearance was typical of a slightly asymmetric micromandibulism with short mandibular bodies and a retruded chin (Figs. 12a,b). Both TMJs were painful on palpation. There was TMJ pain during eating. The occlusion was severely disturbed (Figs. 12c–e). On each side of the maxilla, the first premolar was missing, as well as one lower front tooth. There was bilateral class II occlusion. Occlusion was only possible in the molar regions. At maximum mouth opening there was deviation of the mandible to the left side to a mild degree. Mouth opening was within the normal range. In front of the chin a movable foreign body could be palpated.

Radiographs. *The panoramic view* (Fig. 12f) shows an almost normal shape and structure of the mandible. However, there is a pronounced antegonial notch on the left side while the gonion on the right side is of normal shape. The left ramus is shorter in height than the right and the left condylar process seems shorter and its neck narrower than that on the right. The midline of the chin is slightly to the left.

Tomography of the TMJs (Figs. 12g,h): The left condylar process is much smaller than normal. The condyle and the upper half of its neck shows the typical appearance of secondary sclerosing arthritis, with a very irregularly sclerosed structure. The condylar neck is much shorter and thinner than normal. The right condyle is also smaller than normal but not as much as the left one. Its trabeculation close to the surface is rather dense with irregular translucent areas below the surface. The surface is slightly irregular and sclerotic with a small anterior beak. Its neck is within the range of normal.

The lateral cephalometric view (Fig. 12i) shows the typical skeletal anomaly of “micromandibulism” with open bite. The chin is almost normal in its configuration, but rather short in its vertical height. In front of the chin there is an almost translucent, foreign material implant.

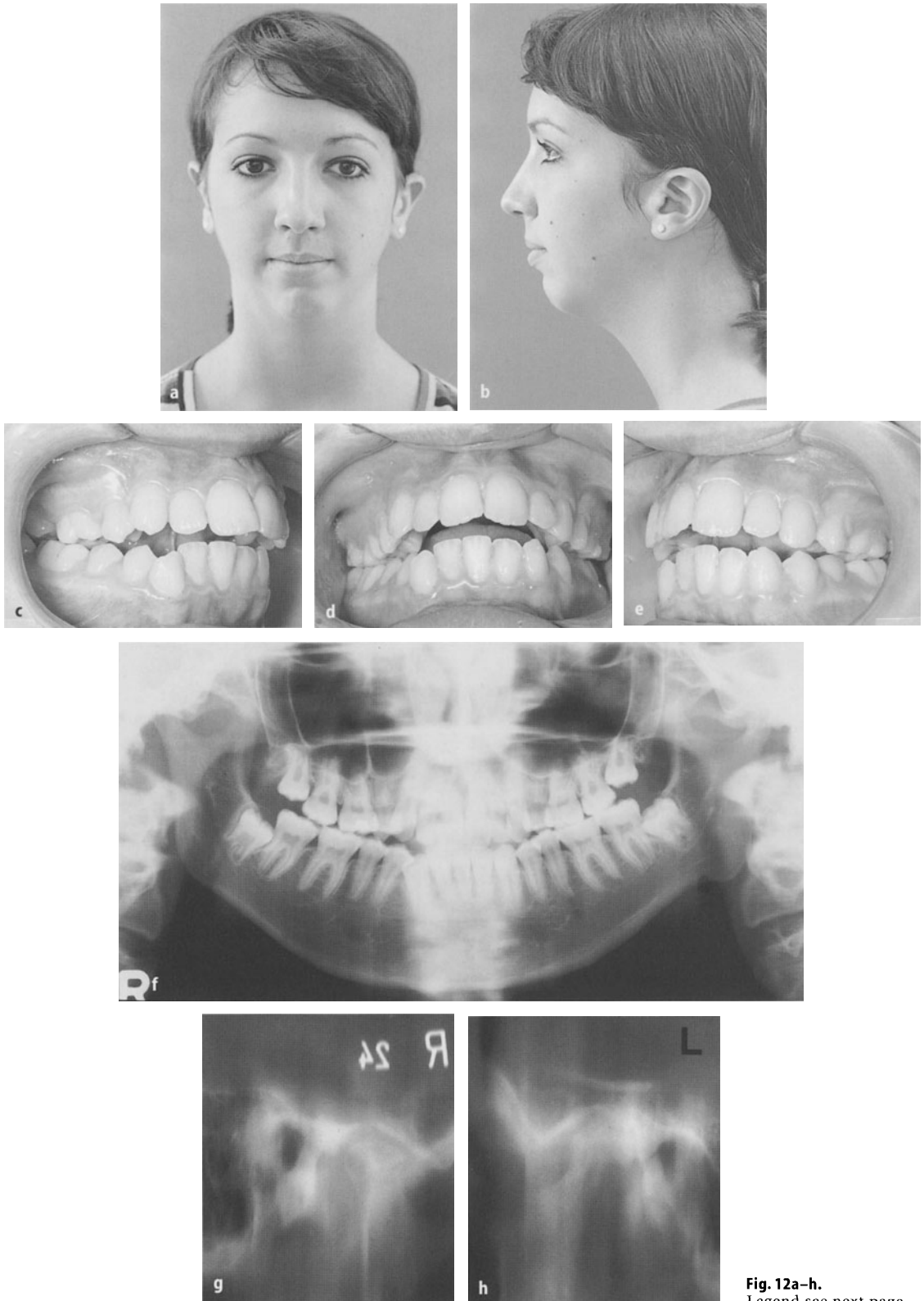


Fig. 12a-h.
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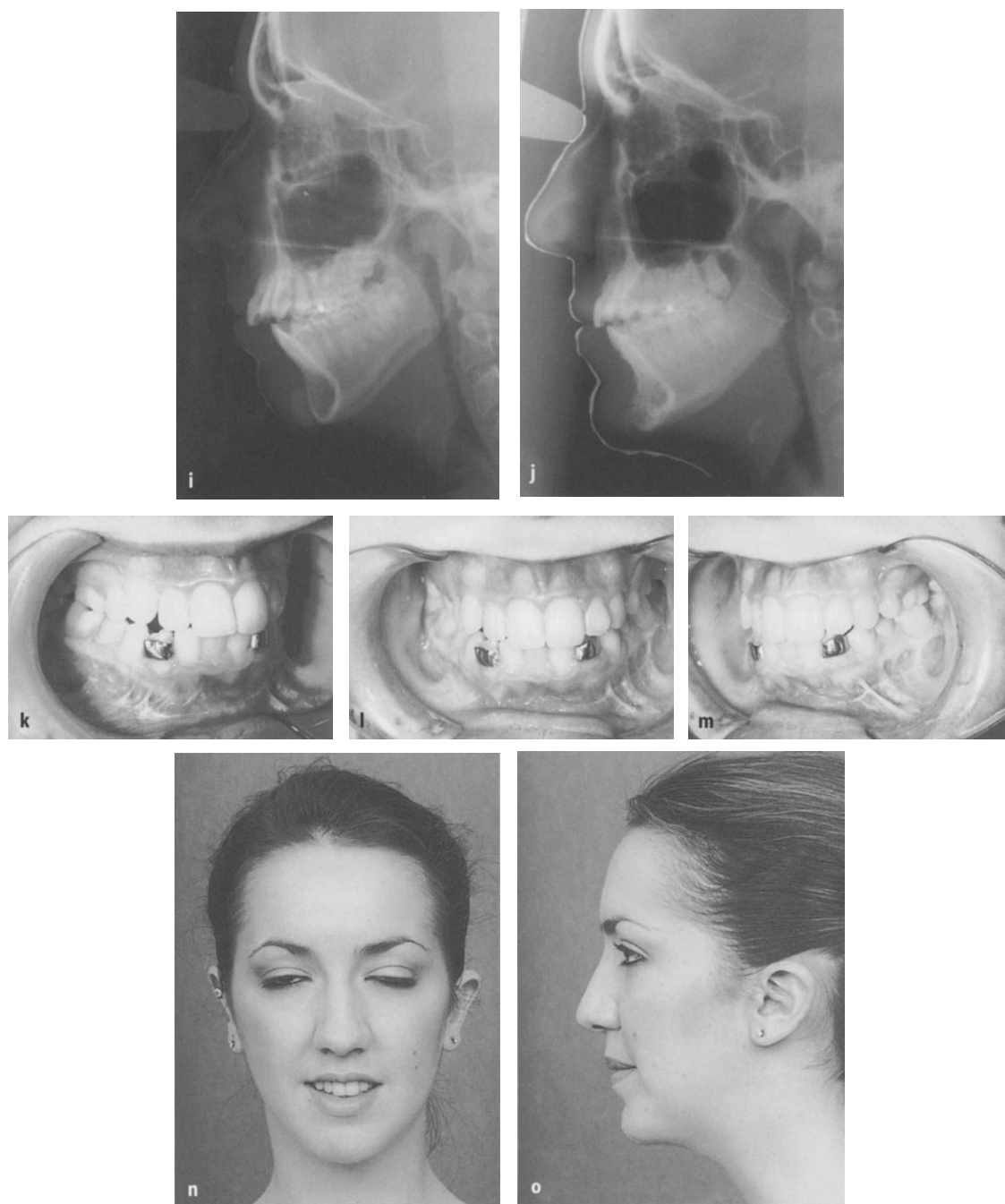


Fig. 12a–o. Case of bilateral asymmetrical condylar hypoactivity due to juvenile arthritis. Patient's presurgical **a** front and **b** profile views showing slight asymmetry of the chin to the left and marked retromandibulism. **c–e** The presurgical occlusion pictures show a circular open bite and the situation after earlier extraction of teeth 14, 24 and 31 with a class II relationship. **f** The panoramic radiograph shows the consequences of earlier condylar hypoactivity, mainly on the left side. **g, h** The TMJ tomograms disclose that both condyles have been affected by arthritis, some time in childhood, the right less severe than the left. **i** The presurgical lateral cephalogram shows clearly the shortness of the mandible and its retroposition with an open bite. The configuration of the chin is normal, but it is short in height (depth). In front of it the silastic chin implant is recognizable. **k–m** Patient's occlusion 6 months after surgical correction. **n** Front and **o** profile appearances. **j** The lateral cephalogram taken at the same time discloses the elongation of the horizontal rami achieved and some calcification of the lyo-cartilage chin onlay. It also discloses the tendency to a slight open bite relapse

Diagnosis. Bilateral asymmetric condylar hypoactivity after arthritis in early childhood.

Treatment Plan. Advancement of the mandible and rotation to close the anterior open bite by the bilateral sagittal splitting of the rami procedure and simultaneous one-step chin sliding advancement after removal of the silastic chin implant. After surgery the patient will be referred to a gnathologist in her home country.

Treatment Performed. The surgery was performed as planned, followed by 6 weeks intermaxillary fixation. Seven months later, slices of lyophilized cartilage were inserted subperiosteally on both sides of the mandible for contour improvement. In addition, a piece of such cartilage was fixed to the chin area in order to increase its prominence further as some of the advanced chin border (not muscle pedicled) had resorbed. Thereafter the patient was referred to specialists in her home country for the necessary treatment of her teeth and occlusion.

Result. The occlusion was as planned (Fig. 12k–m) and so was the facial configuration (Fig. 12n,o). Two and a half years after the correction the occlusion again showed a tendency to a slight open bite in the anterior region as can be recognized on the lateral cephalogram (Fig. 12j).

Discussion. Arthritis of the TMJ during the growth period has a negative influence on the growth of the affected side of the mandible. Depending on the age of the patient and on the severity of the inflammatory process, the negative effect on growth development can vary from the dramatic to the very mild. Although the purely intraarticular inflammatory process has no direct connection with the other part of that hemimandible the development of all sections of it is negatively influenced by it and not only the condyle. This is very interesting and worthwhile mentioning as there is normal mouth opening, contrary to an ankylosis case. It seems as if the so-called “functional matrix” has remarkably lost its influence on mandibular growth through the damage to the condyle. In such a situation the affected condyle acts rather like a brake on the whole hemimandible.

The inflammatory reaction of the various joints can differ quite remarkably from one joint to another as observed, in the large and in the small joints of the extremities as well as in the TMJs. In the extremities the epiphyseal disc takes the responsibility for the growth of that long bone and not the cartilaginous covering of its end which almost looks like a condyle before growth has ceased. The arthritic inflammation of the joints of the long bones obviously does not directly involve that epiphyseal cartilage. In the case of arthritis of the knee joint the epiphyseal cartilage increases in size, resulting in increase in growth stimulation with additional length

of the tibia. However, its calcification will be earlier than normal so that finally there will be no difference in length of the two tibial bones (L. Kaufmann 1998, personal communication). In the growth physiology of the mandible there is no such epiphyseal cartilage and yet the development of the entire hemimandible is involved by the inflammatory damage occurring to the cartilage of the condyle only. The amount of damage and effect on mandibular development depends on the degree of intensity of the inflammation and the developmental stage of the mandible when it happens. It is therefore easily understandable that one side of the mandible may be more disturbed in its growth development than the other. For that reason, we find asymmetric as well as symmetric reduction of mandibular growth which produces either an asymmetric bilateral mandibular hypoplasia (asymmetric micromandibulism) or a symmetric mandibular hypoplasia (symmetrical micromandibulism).

The history of this young lady indicates that she had had various infectious diseases at the age of 9. We must assume that at the same age the infection in her TMJs started. Otherwise the very severe condylar hypoplasia on the left side, already present at the age of 14, would not be explicable. The presurgical orthodontic treatment was not very successful. Regrettably, the postoperative orthodontic treatment was not any better.

Conclusion. Arthritis of the TMJ in early childhood damages the condylar cartilaginous region to any degree between mild and severe depending on the severity of the inflammatory process, the age of the patient and the duration of the active inflammation. Depending on the amount of damage to the growth regulating area, a degree of condylar growth hypoactivity will result with consequent hemimandibular or bilateral hypoplasia. Therefore, corrective surgery should not be performed before growth has ceased and also not before the arthritis has healed. Having learned from cases with traumatically damaged condyles (see case Fig. 26), I would now consider removing the affected condyles as early as possible to allow the functional matrix to stimulate normal mandibular growth or replace the resected condyle with a costo-chondral graft. B. MacIntosh (1985), B. Svensson and R. Adell (1998) have resected the affected condyles in children and implanted costo-chondral grafts. The latter experienced overgrowth of the graft in two-thirds of their cases. It might be worthwhile to implant a rib graft without a costo-chondral junction and leave the growth stimulation to the functional matrix as we did with the iliac crest grafts used for defect reconstruction of the ascending ramus or even larger parts of the mandible in cases of tumour resection including the condyle (see case Fig. 27). In four such cases growth of the graft was experienced congruent with the non-operated side (H. Sailer 1977).

Bilateral Condylar Hypoactivity due to Arthritis in Early Childhood in a Case of Anlage-Induced Micromandibulism (Fig. 13)

Sex and Age. Female, 32 years of age.

Aetiology and History. The mother (Fig. 13a) of the patient had a very short mandible and a retruding chin similar to the anomaly seen in the patient when she was 32 years old.

The panoramic radiograph of the mother, taken at 78 years (Fig. 13b), discloses a similar type and shape of the mandible as seen in case Fig. 11. The horizontal and ascending rami are rather short and the condyles are not at all small or sclerotic as one would have expected if the patient had suffered from juvenile arthritis. They seem to have a rather flat surface as often seen in elderly patients.

The measurement of the tracing of that radiograph revealed equal results in both sides in the length of the horizontal as well as the ascending rami.

The patient herself had an almost normal mandible and chin at the age of 4 years. At that age, the patient suffered from a generalized arthritis of the small joints. But the patient does not remember having had difficulty and pain when opening her mouth. After that age, the mandible and the chin lacked normal growth until the patient's general growth was complete by the age of 18. The first upper premolars were extracted during puberty.

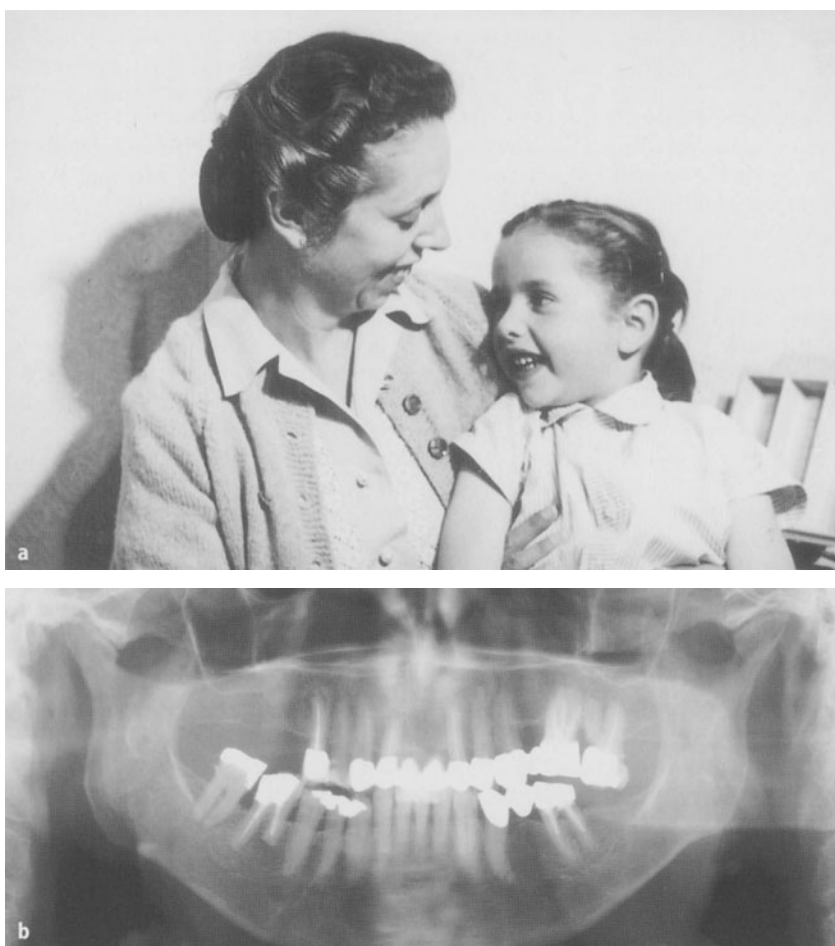


Fig. 13a, b. Bilateral condylar hypoactivity due to arthritis in early childhood in a case of anlage-induced micromandibulism. **a** The patient at the age of 4 years has a normal chin configuration and a normal position of the mandible, while her mother has a typical appearance of micro- and retromandibulism with retrogenia. **b** Mother's panoramic radiograph taken at age 78 shows the same type of mandible as seen in Fig. 11d and also no condylar hypoplasia



Fig. 13c–h. Bilateral condylar hypoactivity due to arthritis in early childhood in a case of anlage-induced micromandibulism. **c** Front and **d** profile appearance of the patient at age 32 years. **e–g** Presurgical occlusion, teeth 14 and 24 are missing. **h** Presurgical panoramic radiograph with typical signs of former bilateral condylar arthritis

Clinical Findings. Symmetrical micromandibulism and retrogenia (Fig. 13c,d). The midline of the chin was in the midline of the face. The mandible was very narrow. In the maxilla, a bicuspid was missing on each side after extraction in childhood (Fig. 13e–g). Otherwise there was a full dentition in class II occlusion. There was no shift of the midline of the lower teeth to either side. Excessive mouth opening was possible.

Radiographs. The panoramic view (Fig. 13h) reveals a mandible with short ascending rami and small condyles. Both rami look vertical. Bilaterally, there is a clear but not very deep antegonial notch, moreso on the right side than on the left. The latter is rather flat. Compared with normal there is some lack of height of the body in the posterior molar region. The lower front teeth are tilted towards the left side. Bilaterally there is a class II occlusion.

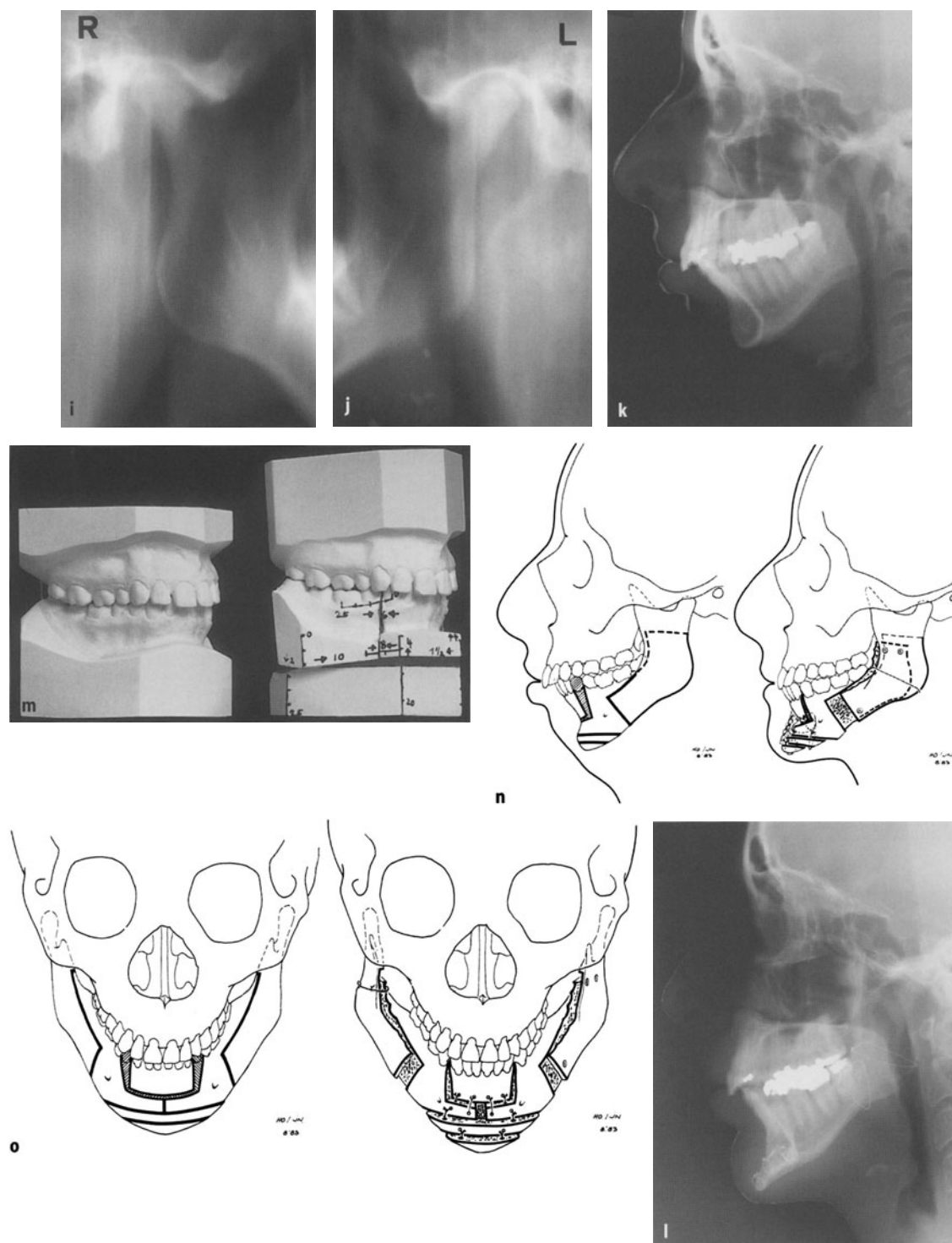


Fig. 13j–o. Bilateral condylar hypoactivity due to arthritis in early childhood in a case of anlage-induced micromandibulism. **i, j** The TMJ tomograms show on both sides the typical signs of arthritis in early childhood. **k** The preoperative lateral cephalogram discloses that both the horizontal as well as the ascending rami are too short. **l** The postoperative lateral cephalogram taken 2 years and 3 months after surgery, shows the remarkable amount of elongation of the horizontal rami. **m** Model-operation-planning. **n, o** Schematic drawing of the author's (1988) one stage procedure for correcting a micro-retromandibulism case with retrogenia, in front and lateral views

TMJ-ramus tomographs (Fig. 13i,j): Both condyles are smaller in size than normal and deformed in their shape, with narrow condylar necks and sclerosed trabeculae and surface. The right ascending ramus shows to a mild degree the shape seen after a juvenile arthritis, with a rather shallow antegonial notch and concavity of the posterior border of the ascending ramus. The shape of the left ascending ramus is closer to normal but it also to a certain extent demonstrates the influence of the juvenile inflammatory damage to the condyle.

The lateral cephalometric view (Fig. 13k) shows short ascending rami and mandibular bodies and chin. The angle of the mandible is almost normal in shape. There is a slight, rather shallow antegonial notch; no abnormal trabeculation structure is visible.

Diagnosis. Postarthritic bilateral condylar hypoactivity in a case of hereditary micromandibulism.

Treatment Plan. The treatment plan has to be made according to the basic principles: *“Leave only as many teeth in the alveolar process as can be positioned in their*

proper angulation to their respective jaw base. Alternatively, elongate the jaw base within the tooth bearing area” (Principle Nr. 40) and *“In a case of micromandibulism, the mandible not only needs elongation but also widening”* (Principle Nr. 38). Applying these two principles, the model operation (Fig. 13m) was used to find out what occlusion could be achieved when the first premolar on each side of the mandible was removed and the anterior alveolar segment repositioned posteriorly, bringing the lower anterior teeth into proper angulation to the jaw base. For this it would be necessary to widen the mandibular arch. Only following the extraction of the first premolars does it become possible to advance the mandible using a long-surface, bilateral ramus-body splitting procedure. In addition to the widening of the mandibular body, a double step chin advancement has to be performed for the necessary elongation of the mandible in its anterior part (see Fig. 13n,o).

Treatment Performed. The surgery was executed according to the planning, followed by 6 weeks intermaxillary fixation. Good occlusion with a good anteface

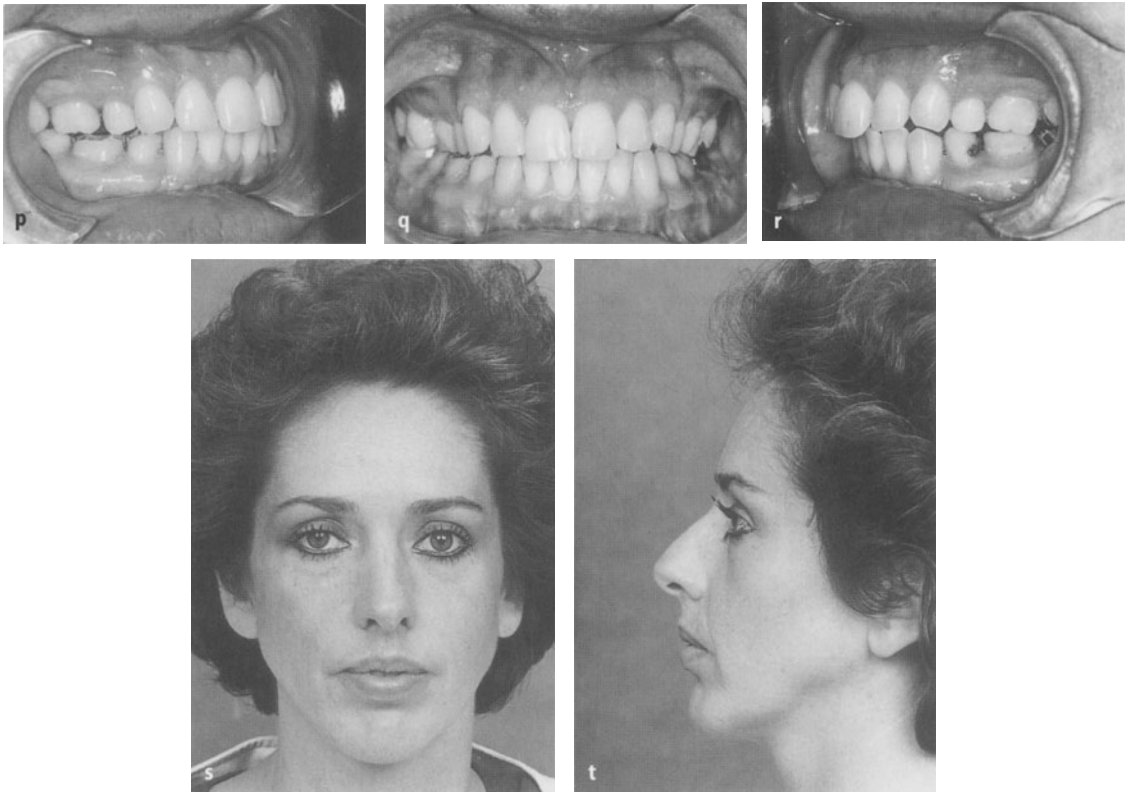


Fig. 13p–t. Bilateral condylar hypoactivity due to arthritis in early childhood in a case of anlage-induced micromandibulism. **p–r** Occlusion 2 years after surgery, without any orthodontic treatment. **s,t** Patient's frontal and lateral appearance 2 years after surgery. The suggested correction of the lateral depressions, a consequence of the large amount of forward advancement of the mandible, was not deemed necessary by the patient

profile and some widening of the mandible was achieved. Two years after surgery there was no change in occlusion or profile (Fig. 13l,p-t) and the same still applied 13 years later.

Discussion. In cases of inherited or anlage-induced micromandibulism, the condyles are within the normal range in size and shape. The mandible is symmetrical. There is no deviation of the midline of the lower teeth or chin. The shape of the mandible and its gonial angles are almost normal. There is no pronounced antegonial notch or posterior ramus concavity. This specific case obviously has a hereditary origin, but in addition, there was some influence of juvenile TMJ arthritis. If it were of inherited origin only, the specific signs of postarthritic shape and sclerosed structure of the condyles would not be present. If it were of arthritic origin alone, the specific postarthritic shape would have been more pronounced, particularly in the pregonial area. There-

fore, I believe that this case is a mixture of anlage-induced micromandibulism and postarthritically-influenced condylar hypoactivity.

Conclusion. Inherited or anlage-induced micromandibulism is due to genetic programming of the shape of the mandible with condyles close to normal in size and structure. Additional postarthritic condylar hypoactivity changes the shape and structure of the mandible towards the postarthritic type. Due to the dual aetiology of the lack of mandibular growth, the lack of development is more pronounced than when only one of them is the cause. The radiographs of the condyles permit a clear diagnosis that an inflammatory cause also existed.

However, if this severe birdface appearance were due to inflammatory aetiology only the condyles should be much more destroyed as is true in the case of Fig. 12 on the affected side and also in case of Fig. 14.

Bilateral Condylar Hypoactivity due to Untreated Bilateral Luxation of the Mandible in Infancy (Fig. 14)

Sex and Age. Female, 20 years of age.

Aetiology and History. Unknown. There is no relevant family history. The pregnancy and labour had been normal, in particular there was no forceps delivery. Breast feeding was normal. Between the age of 5 and 6 years the girl suffered from measles, varicella when she was 7 years old, and at the age of 10 years, infectious parotitis. Already in early childhood and before her infectious diseases her chin seemed not to have developed as far forwards as one would normally expect. But since there are always children with a certain lack of mandibular forward growth it was not considered to be unusual. When the girl was 12 years old she was taken to an orthodontist because her mother had noticed she had an open bite.

Clinical Findings. Typical symmetrical bird face appearance with normal position of the upper and lower lips, but almost no chin (Fig. 14a,b). Maximum spontaneous incisor opening of 33 mm (Fig. 14c), then difficulty in breathing. Forced opening up to 40 mm possible, but then breathing is blocked. Lateral deviation of the mandible to both sides is slightly less than normal. No condylar movement palpable in both preauricular areas. Class II occlusion by half a premolar. Severely protruding upper and lower anterior teeth (Fig. 14d-f).

Radiographs. The panoramic view in the closed position (Fig. 14.7) shows short horizontal and ascending rami with deep antegonial notches and empty glenoid fossae.

TMJ-ramus tomograms in the closed position (Fig. 14h): on both sides there is no real condyle or condylar neck. The upper part of the posterior section of the ascending ramus shows, on both sides, a slight nodule-like thickening, probably the remaining rest of the former condyles. In the closed mouth position on the left side the nodule is placed in front of the area where the articular prominence should be and on the right side it sits exactly on that area. The posterior borders of the ascending rami are directed forwards as in adult cases of mandibular luxation and not towards the glenoid fossa as one would expect in a posttraumatic ankylosis case (see Fig. 22h).

The lateral cephalogram (Fig. 14i) discloses the typical shape of a micromandibulism with micro- and retrogenia, very deep antegonial notches and bimaxillary teeth protrusion.

Diagnosis. Micromandibulism and micro-retrogenia due to bilateral condylar hypoactivity probably as a consequence of undiagnosed bilateral luxation of the mandible, occurring in very early childhood, resulting in destruction of both condyles.

Treatment Plan. Standard correction procedure of symmetrical birdface appearance when presurgical orthodontics is not available (H. Obwegeser 1988b) (see Fig. 13m,n). With the help of the model operation the

kind of occlusion was determined which could be achieved by the use of the standard procedure for severe birdface correction. As the result of this the following treatment plan was accepted: Extraction of the first premolar on each side of the mandible. Next mobilization of the lower alveolar segment containing the four incisor teeth and the canines for posterior repositioning in order to bring these teeth into proper angulation to the base of the mandible and to obtain anterior space to advance the mandible. Then the prominence of the chin will be detached leaving it pedicled on the lingual musculature for advancement followed by a bilateral long-surface sagittal splitting procedure (H. Obwegeser 1968a) for mandibular advancement. Next the mandible will be divided in the chin area (symphysotomy) (Fig. 14k) permitting the necessary widening of the arch of the mandibular bodies in order to reposition the anterior alveolar segment (Fig. 14l). After that, fixation of all the segments in the planned new positions (at that time achieved with wire osteosynthesis). The gap in the symphysis region will be filled with small pieces of bank cancellous bone (Fig. 14m), 6 weeks intermaxillary fixation will allow bony union of all parts in the new position.

At a second session, an additional sliding genioplasty will be planned, to bring the chin prominence further forward and slices of lyophilized cartilage will be on-layed subperiostally to increase the width of the mandibular contour.

Actual Treatment and Result. As the anaesthetist was unable to intubate the patient, a tracheotomy was performed. Otherwise the operation was executed as planned, however, with only a one-step advancement of the chin because of insufficient chin height for a two-step advancement. In a second operation the chin was further advanced by a sliding osteotomy, and slices of subperiostally implanted lyophilized cartilage were used to improve the width contour of the mandible, as originally planned.

The surgical correction produced a very acceptable facial skeletal form (Fig. 14j) with a good occlusion without any orthodontic treatment (Fig. 14p-r). There was a good profile line but still a rather narrow mandible (Figs. 14n,o).

Discussion. The possible aetiology of the severe lack of mandibular growth in this young lady invites discus-

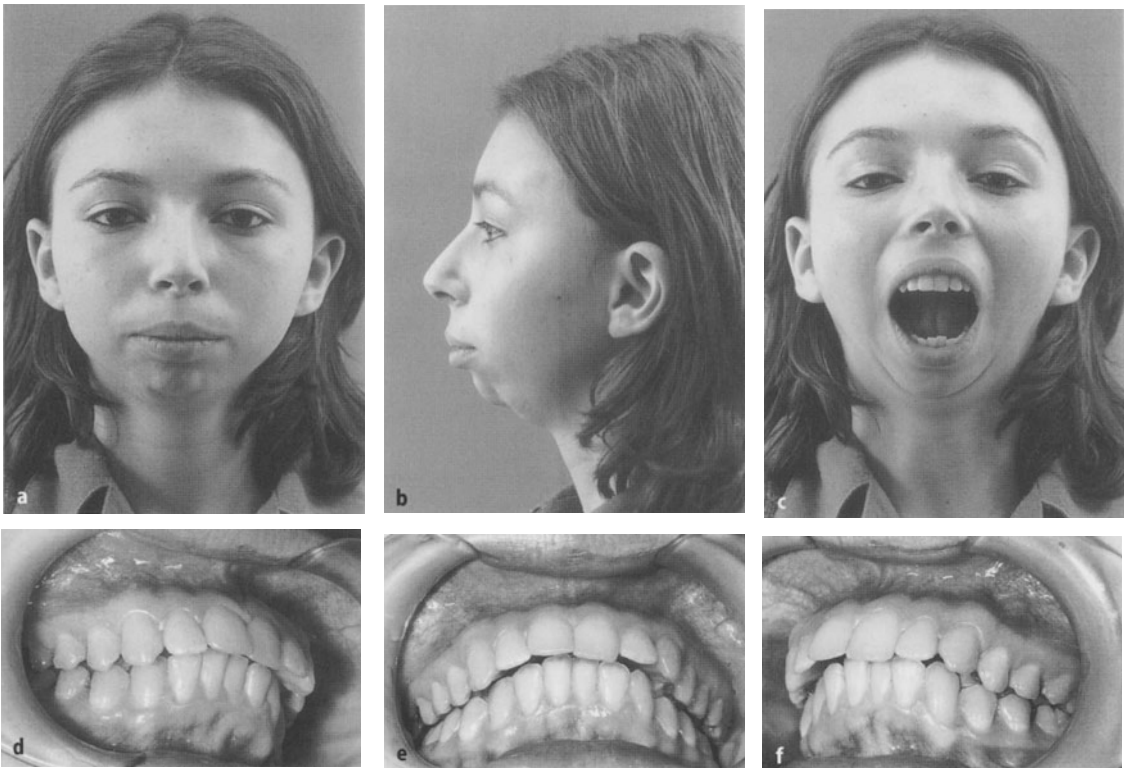


Fig. 14a-f. Case of severe symmetrical microretromandibulism with retro- and hypogenia, due to bilateral condylar destruction as a consequence of untreated bilateral luxation of the mandible in infancy. **a-c** Patient's front and profile appearances and reduced mouth opening before surgery. **d-f** Occlusal situation before surgery

sion. According to the shape of the mandible and the lack of posterior downward growth of the maxilla, the cause of the anomaly must have affected the condyles very early in life. Until that event, the mandible will have

developed normally, whatever the cause might have been. So, depending on the age of the patient when it happened we may find clear signs of normal forward development.

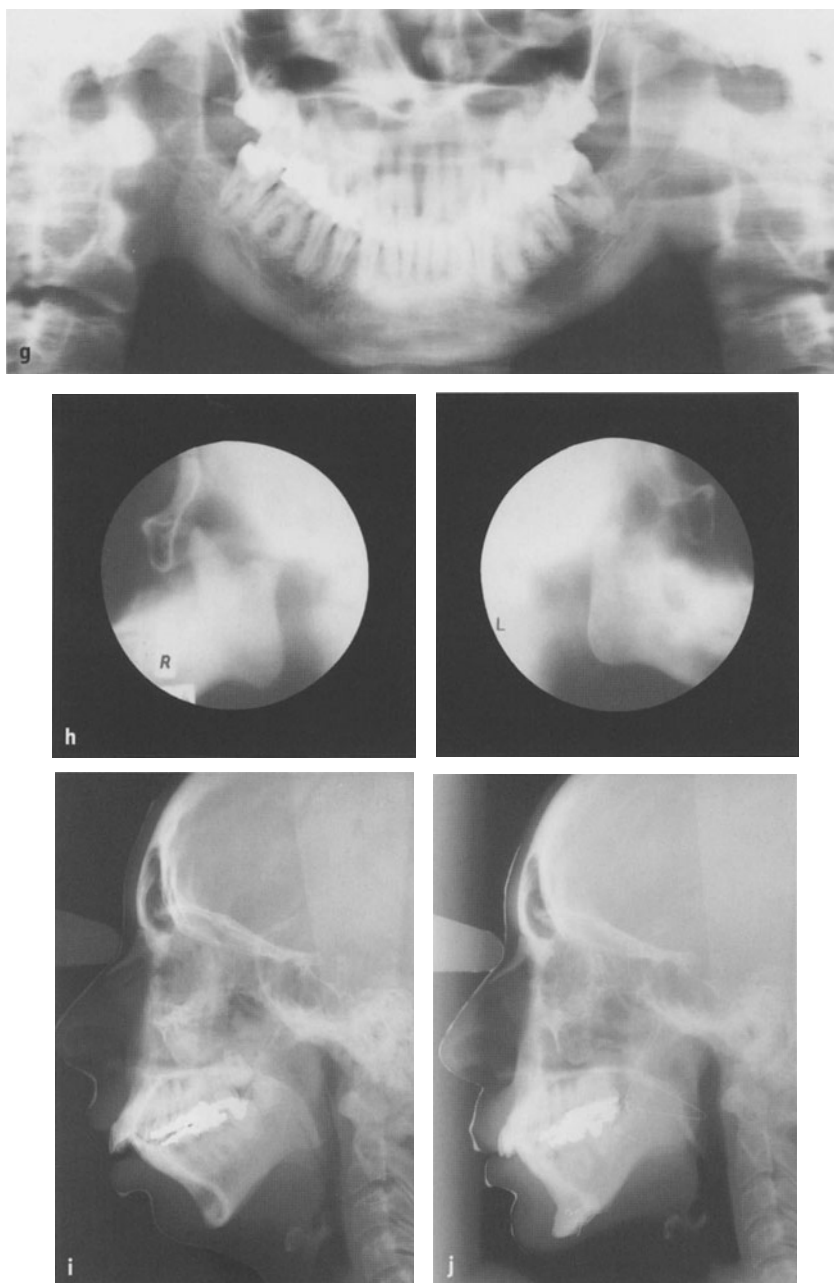


Fig. 14g-j. Case of severe symmetrical microretromandibulism with retro- and hypogenia, due to bilateral condylar destruction as a consequence of untreated bilateral luxation of the mandible in infancy. **g** Panoramic radiograph in closed position demonstrating the very small mandible with little remnants of the condyle only, and with the posterior border of the rami directed toward the articular prominence on the right side and in front of it on the left side. **h** Right and left TMJ-ramus tomograms showing shortness of ramus length directed towards and in front of the articular eminence with little remnants of the condyles and their neck. **i, j** The lateral cephalograms before and 2 years after final surgery demonstrate the shortness and the achieved length of the horizontal rami and prominence of the chin and the change in the angulation of the lower front teeth

G. Rabey (1977/1978) published a very interesting paper on this subject, based on four personal cases and four reported in the literature. He believes cases like this are due to a condylolysis. That is, of course, possible. But it was our experience that such an almost complete condylolysis happens after severe purulent destruction only, and not after a mild or even severe arthritic infection or after trauma either. I have never seen such a massive destruction by infection with subsequent resorption of the condyle bilaterally. And when bilateral, the amount of damage to the condyles differed. Whether the damage to the condyles is due to a purulent infection or an inflam-

matory process of the condyles or any other aetiology, the results may be very similar. There will be a partial or complete destruction or resorption of the condyle. But whatever the cause, to me it is incomprehensible as to why the posterior border of the condyle should end up directed towards the articular prominence or even in front of it after condylolysis due to arthritis.

I can imagine that bilateral luxation of the mandible can easily be overlooked by the child's parents, just as a paediatrician can overlook bilateral condylar fractures. If there is bilateral luxation of the mandible there is no asymmetry to draw the parents' attention and it will not

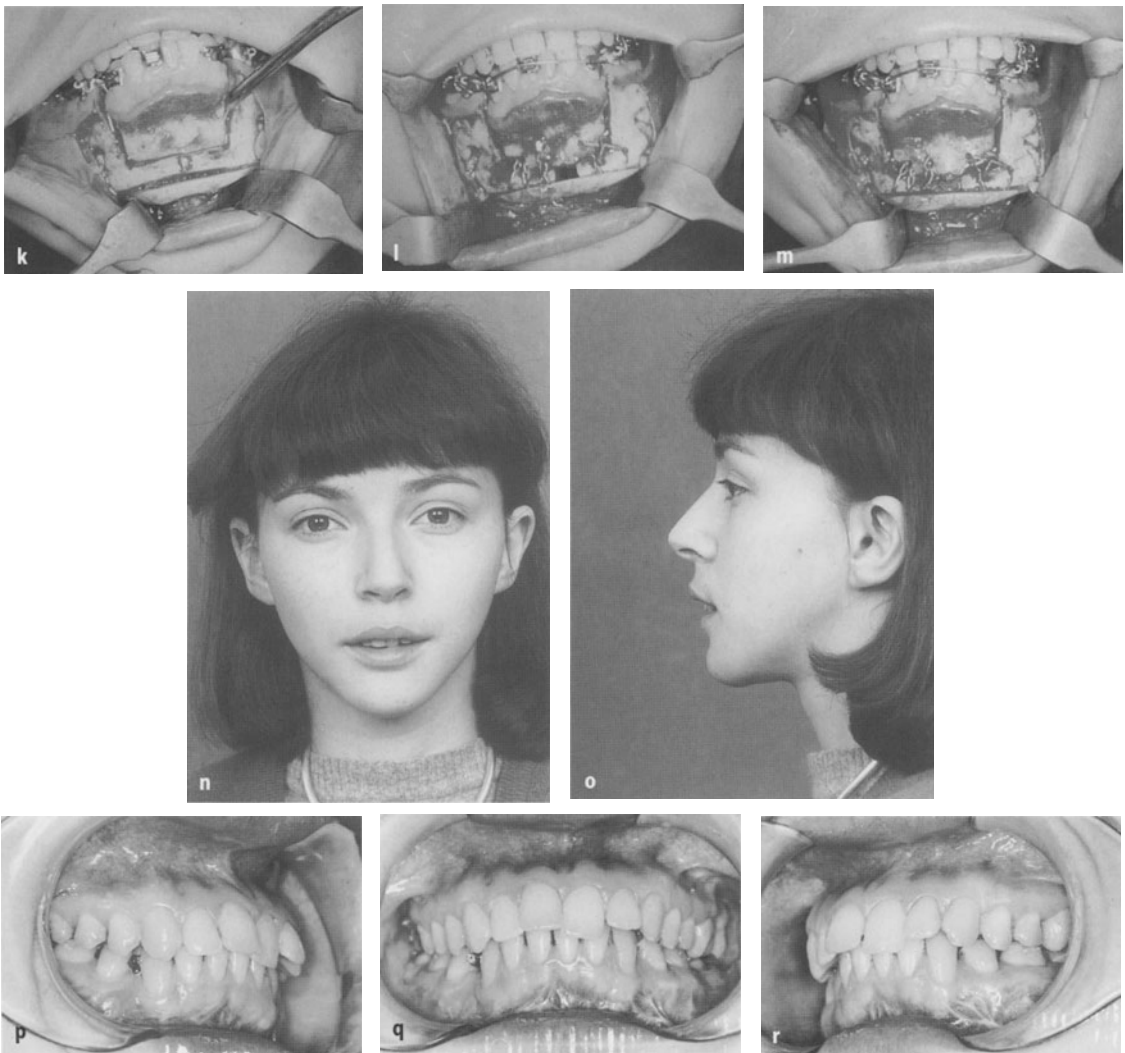


Fig. 14k-r. Case of severe symmetrical microretromandibulism with retro- and hypogenia, due to bilateral condylar destruction as a consequence of untreated bilateral luxation of the mandible in infancy. **k-m** Demonstrate the situation of the widening of the narrow mandibular arch and the protrusion of the lower front teeth during operation stage I. **n, o** Show the patient's appearance 2 years after operation No. 2. The mandible has come forward into proper position and the height of the lower facial third has increased remarkably. **p-r** The patient's occlusion 2 years after surgery with shortening of the lower dental arch by one bicuspid on each side, in order to be able to widen it and advance the mandible further forward

take long before the child can close its mouth again, maybe a little further anteriorly. I have also seen that happen in an adult who had a bilateral mandibular luxation for almost 30 years, acquired between 20 and 30 years of age. As he was in a nursery for mentally retarded patients, the condition was not noticed. In early childhood and even more in infancy, the condyles will be resorbed because of jaw function in the new position. Only in such a situation can I understand the anterior direction of the ascending ramus, which is not seen in congenital aplasia of the condyle (see case Fig. 15). Although this patient had measles between age 5–6 years this probably had nothing to do with the ascending ramus ending up in a position seen with bilateral luxation. If the situation were due to a bilateral condylar fracture an ankylosis would develop rather than resorption of the condyles with fairly good mouth opening and still the posterior border of the ascending ramus remaining directed towards the glenoid fossa. I can easily imagine that luxated condyles will become resorbed when the luxation happens in infancy. Since there is still some sort of remnant of the former condyle-neck anatomy present and in particular since the ascending rami are in the same direction as in a bilateral mandibular luxation situation, I diagnose this case as being a consequence of very early, non-reduced bilateral luxation of the mandible and not as a consequence of bilateral condylar fracture or of bilateral TMJ infection or arthritis.

In my opinion, one further point favours that aetiology: it is the very pronounced antegonial notch and the lack of posterior downward growth of the maxilla, both clearly seen in the presurgical lateral cephalogram. This happens to such an extent only when the lack of upward growth of the ascending ramus occurs very early, before the age of 5–6 years.

So, we may call also this case a condylolysis, but rather as a result of the bilateral luxation of the mandible during the first months or years of life than after a general infectious disease at the age of 5–6 years.

Conclusion. Non-reduced bilateral luxation of the mandible, occurring in infancy, may result in destruction of the condyles with consequent severe condylar hypoactivity leading to extreme lack of mandibular growth, yet with almost normal mouth opening. The severely damaged condyles seem to eliminate the functional matrix as a growth stimulus. In our records we have four similar cases with the same diagnosis.

10.1.2 Unilateral Condylar Hypoactivity

While we assume the existence of two growth regulators in cases of hyperactivity, one stimulating growth in length and the other production of bone mass, in the cases with hypoactivity we do not know two so typical-

ly and different forms resulting from damage to the condylar cartilage zone. Condylar hypoactivity always results in an ipsilateral hemimandibular hypoplasia. The form of the mandibular half in hypoplasia of the condyle is characterized by a shortness of the ascending ramus and body of the mandible on the relevant side. The gonial angle is almost rectangular, always with a notch in front of it. The mandibular body is smaller in height than a normal mandible and the ascending ramus is remarkably thinner which can cause some difficulty to the inexperienced surgeon when executing a sagittal splitting procedure. On top of the thin ascending ramus sits a slim condylar neck with a more or less small condylar head. In the radiographs it can vary from an almost osteolytic translucency up to sclerotic condensation with an irregular surface. Naturally, it can become very difficult to say, in borderline cases, that a condyle is too small or too large. The resulting variability in size and form of the condylar head is very great. Clearly during mandibular growth, hypoplastic condyles always lead to a hypoplasia of the same mandibular half.

Whenever a diminution of the condyle occurs after growth has ceased, either as a result of inflammation or trauma, the size of the pertinent mandibular half will not change. Contrary to hyperactivity this indicates that damage to the condylar cartilage zone does not have a negative influence after growth has ceased. Hypoplasia of one half of the mandible leads to its lateralisation to the abnormal side. Because of its shortness, the midline of the mandible is deviated to the abnormal side and also the midline of the lower front teeth and the midline of the chin. These signs are also typical for the elongation of a mandibular half in the case of H.E. Consequently, unilateral mandibular hypoplasia can easily be misdiagnosed as an unilateral contralateral H.E.

Since hypoplasia of one half of the mandible can develop during embryonic or postnatal growth, it will automatically have a negative influence on the growth and development of the maxilla. The maxilla cannot grow downwards normally on the affected side. As a consequence, an oblique occlusal plane will always develop in unilateral hypoplasia cases. That oblique occlusal plane, accompanied by an oblique higher lip commissure on the hypoplastic side, is the decisive differential diagnostic sign against H.E. which cannot produce an oblique occlusal plane as it produces a lateralisation of the mandible only, without a shortening or increase in ascending ramus height seen in hemimandibular hypoplasia or in H.H. on the opposite side. The latter cannot be confused with a unilateral hemimandibular hypoplasia because of the entirely different shape on the affected side.

Congenital Unilateral Condylar Hypoactivity

Those cases of condylar hypo- or aplasia which are congenital forms of condylar hypoactivity are more suit-

able for studying secondary disturbances of growth than those which have been produced by external adverse influences causing secondary hypoactivity after normal primary activity.

Naturally, it would be desirable to be able to evaluate and follow up a greater number of cases of each category. That requires a large patient population and a well documented follow up over 20 years. Centres for the study of facial growth should be able to give us the answer.

Unilateral Congenital Aplasia of the Condyle (Fig. 15)

Sex and Age. Male, 7 years of age.

Aetiology and History. Congenital. Facial asymmetry was not noticeable soon after birth. The boy had no difficulty in eating or speaking. After eruption of his deciduous teeth a slight crossbite became obvious. The boy was therefore referred to our clinic. In the family history there was no known similar anomaly.

Clinical Findings. Slight facial asymmetry with flatness of the right cheek. The left side of the face has rather less

vertical height than the right. The left lip corner is a little higher and more lateral than the right. The left ear is set a bit lower than the right. The chin deviates slightly to the left. When opening the mouth there is severe deviation of the mandible to the left (Fig. 15a–c). There is a complete deciduous dentition, in crossbite to the left by the width of one lower incisor. There is only a very slightly oblique occlusal plane due to vertical shortness of the maxilla and mandible on the affected side (Fig. 15d).

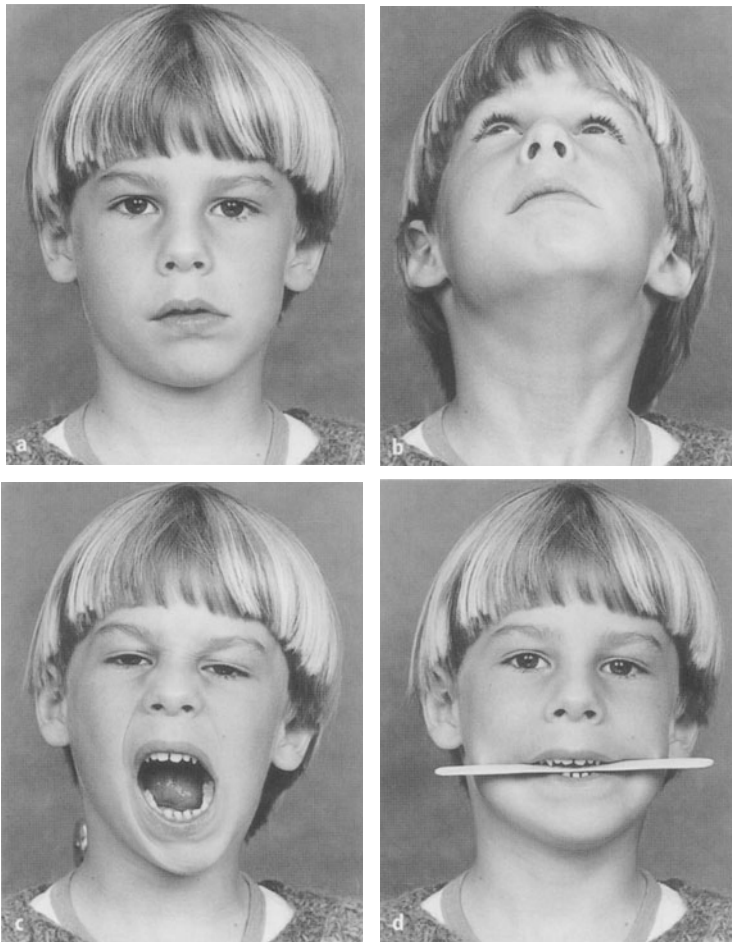


Fig. 15a–d

Radiographs. *Panoramic view* (Fig. 15e): normal shape and structure of right side of mandible. The midline of the chin is slightly to the left. The height of the left body of the mandible is reduced, although not very markedly. The left angle is almost rectangular, but rounded. There is no real antegonial notch, but a flat depression instead. The left ascending ramus is slightly shorter than the right and the coronoid process is within the normal range in shape and length. There is a broad stump instead of a condylar neck, but there is no

actual condyle. Instead of a condyle the stump ends in a broad rounded surface. The normally curved semilunar notch has a triangular shape. There is something akin to a flat glenoid fossa but no real articular eminence.

TMJ tomographs: They are of such poor quality that they are worthless.

Mandible p.a. at maximum opening: There is a remarkable asymmetry of the mandible due to vertical shortness of the left side because of the absent condylar neck and head.

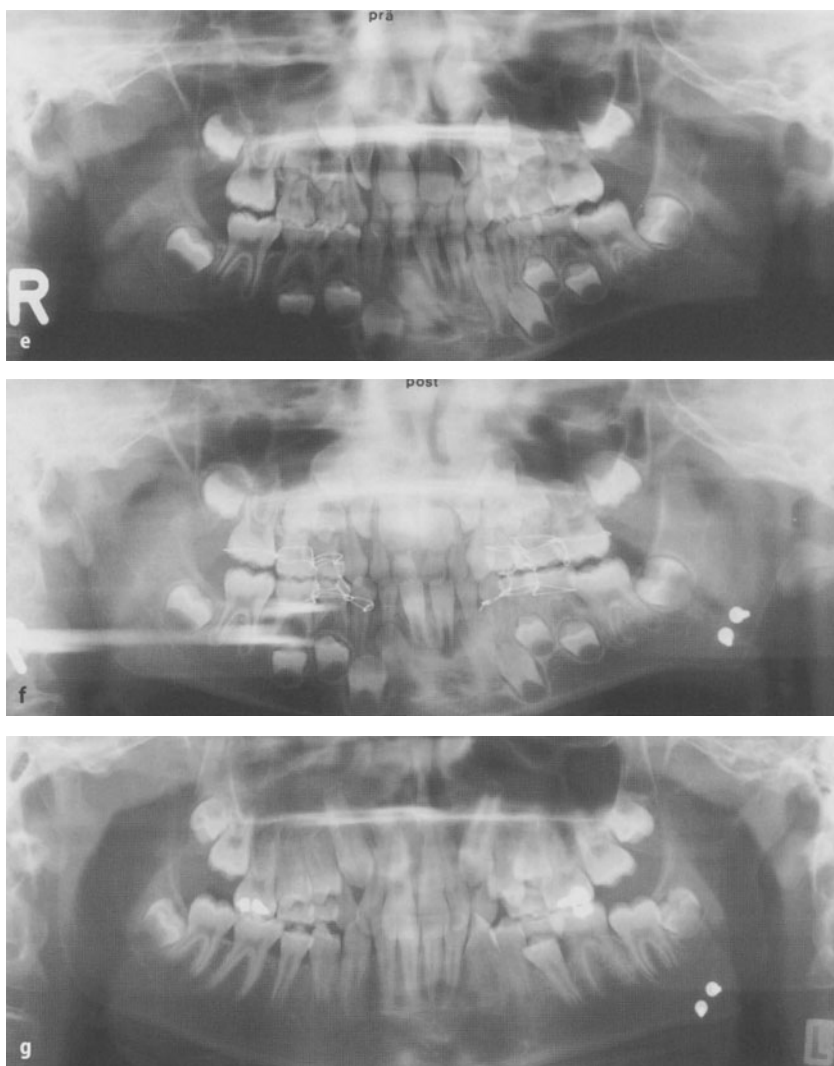


Fig. 15a–g. Unilateral congenital aplasia of the condyle. **a–d** Patient's appearance at the age of 7 years with chin deviation, reduced facial height on the affected left side, severe deviation of the mandible at maximum mouth opening, but little lack of downward growth of the maxilla. **e** Panoramic radiograph discloses a normal mandible on the right side. The left side is lacking in height in the body and the ascending ramus. There is an almost rectangular gonion angle with a flat depression in front of it, but there is no condyle present. The neck of the condylar process is like a broad stump, covered by a sclerotic surface. **f, g** Panoramic radiograph after reconstruction of the missing condyle by a costo-chondral graft, soon after operation and 6 years later

Provisional Diagnosis. Congenital condylar aplasia on the left side.

Treatment Plan. Costo-chondral graft insertion for reconstruction of the absent condyle to provide growth of the ascending ramus and correction of the crossbite.

Actual Treatment and Result (by M. Farmand M.D., D.M.D., then a senior surgeon at my clinic). At the age of 7 years and 1 month, insertion of a costo-chondral graft,

via the oral route with an additional retrotragal TMJ approach, was performed (Fig. 15f). No biopsy was taken from the stump.

Six years later, at the age of 13 (Fig. 15g), the costo-chondral graft has developed into a well-shaped neck and condyle. The previously existing height difference between the bodies of the two mandibular halves has disappeared. The antegonial depression is gone. The triangular shape of the semilunar notch has changed to normal. There is now only a pronounced depression be-



Fig. 15h–j. Unilateral congenital aplasia of the condyle. P.a. projection of mandible in the open mouth position **h** before, **i** 6 months and **j** again 6 1/2 years after surgery, demonstrating the result of the growth of the costo-chondral graft

low the base of the condylar neck at the posterior border of the ascending ramus. The maxilla is grown down to a normal level and the occlusal plane has become horizontal.

The shape of the mandible before surgery and 6 months after reconstruction of the deficient left ramus length, then very asymmetrical, has developed to almost normal size and full symmetry within 6 years (Fig. 15h–j).

The boy now has a symmetrical face and an almost perfect occlusion and normal mouth opening, with very little deviation (Fig. 15k–p).

Histology. Unfortunately no biopsy was taken from the bone stump.

Discussion. In spite of a complete congenital absence of a condyle, a mandible of almost normal shape and size developed on the affected side. The mandibular hypoplasia resulting from the fact that there is no condyle is rather minimal. It is mainly the lack of height of the left ascending ramus which produces the obvious asymmetry and is caused by the absence of the condyle and neck. The lack of height of the body of the mandible is almost minimal and much less than observed in cases of

hemimandibular hypoplasia due to adverse postnatal influences such as trauma or osteomyelitis.

Whether or not the upper surface of the existing stump of the ramus has a cartilaginous layer similar to the condyle, we do not know. Unfortunately no biopsy was taken during surgery.

After the insertion of a costo-chondral graft at the age of 7, within 6 years an almost normal mandible resulted with equal body height on each side and normal mouth opening with almost no deviation and a perfect occlusion.

Conclusion. The congenital absence of a condyle appearing as a single anomaly, not within a syndrome, does not produce a real hemimandibular hypoplasia although it would be expected to do so. However, we do not know whether a similar cartilaginous layer as in a normal condyle exists on the bone stump or not. Therefore, we do not know if a real condylar hypoactivity is present in such a case. It definitely does not act as a growth inhibitor as a severely damaged condyle would do. The early insertion of a costo-chondral graft seemed to eliminate the consequences of the missing condyle, anatomically as well as functionally.

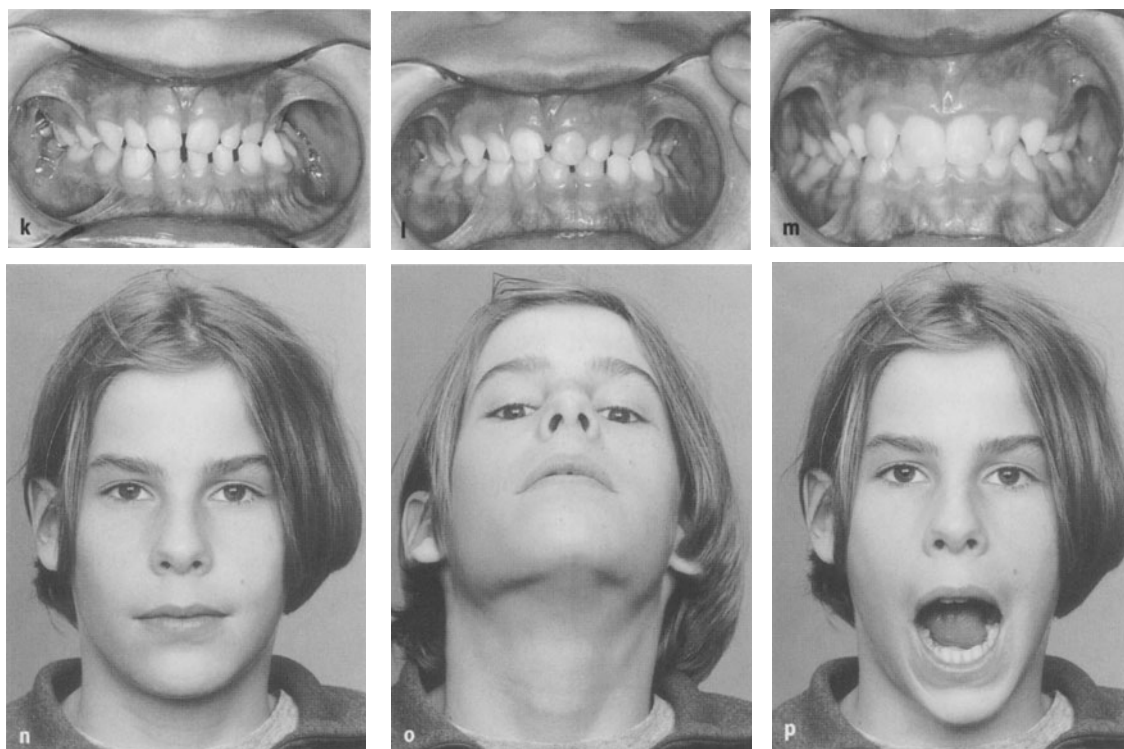


Fig. 15k–p. Unilateral congenital aplasia of the condyle. Occlusion **k** 2 years before, **l** immediately after and **m** 6 1/2 years after reconstruction of the missing condyle. **n–p** Patient's appearance 6 1/2 years after insertion of a costo-chondral transplant via the oral route with an additional TMJ-approach

Unilateral Congenital Hypoplasia of the Condyle¹ (Fig. 16)

Sex and Age. Female, 12 years and 5 months.

Aetiology and History. This girl from outside Switzerland was brought by her parents to Prof. Paul Stöckli, Head of the Department of Orthodontics and Paedodontics of the Zürich Dental School, for evaluation and treatment planning because of an increasing, slight facial asymmetry with an oblique occlusal plane. There were no radiographs from earlier years. However, P. Stöckli was able to obtain from the parents front view photographs of the girl at 1, 4, 5, 8 and 10 years of age (Fig. 16a–e). By marking the facial midline he could clearly demonstrate that already at the age of 1 year there existed a slight hypoplasia of the right side when compared with the left, which gradually increased with

age. In addition the prominence of the chin moved slightly towards the hypoplastic side. There was no history of facial trauma or rheumatoid disease or any other postnatal damage to the right TMJ. The family history was also negative.

Clinical Findings. At the age of 12 years and 5 months there is a slight vertical and horizontal hypoplasia of the right lower face including the zygomatic area, with deviation of the chin to the right side and an oblique labial commissure with the right corner placed more distally and higher than on the left side. This is also true for the right nostril but to a very mild degree only (Fig. 16f).

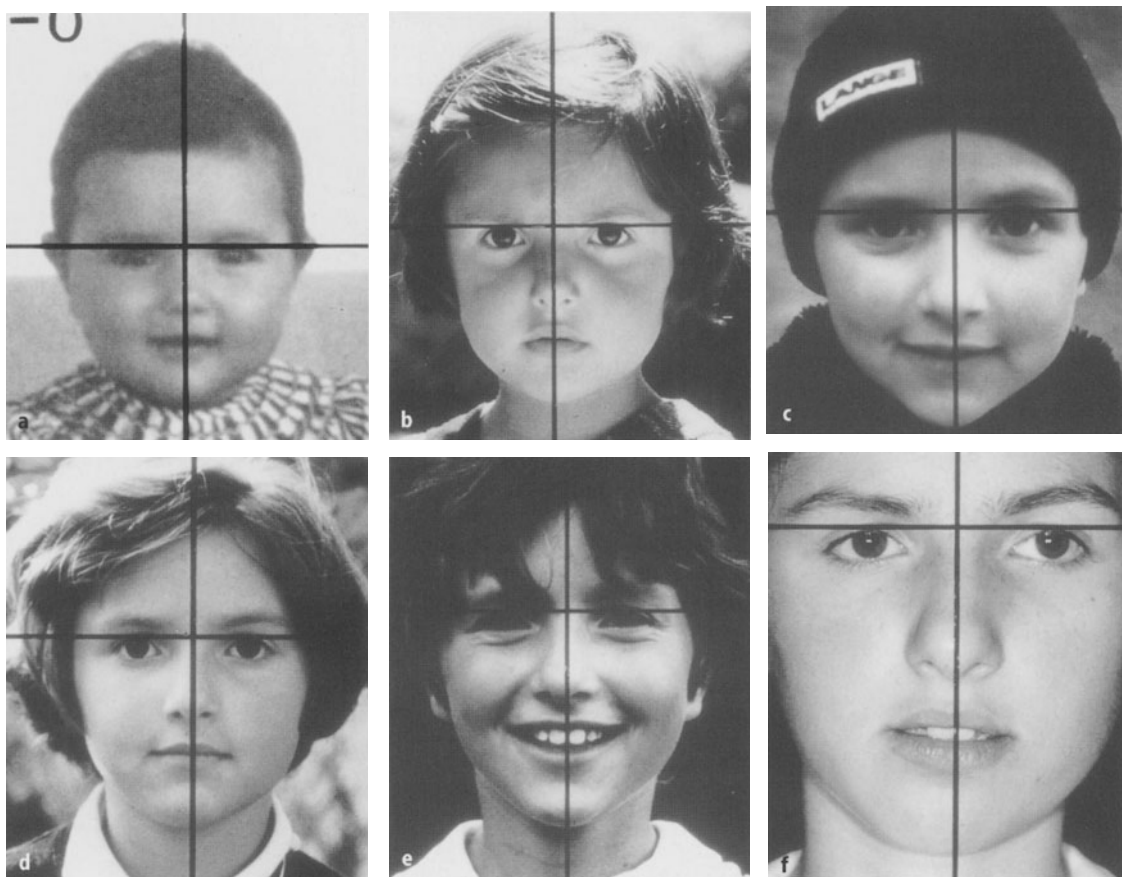


Fig. 16a–f. Case of unilateral congenital hypoplasia of the condyle. **a–f** The girl's facial asymmetry in the front view from age of 1, 4, 5, 8, 10 and 12 years, evaluated by P. Stöckli

¹ I am very grateful to my friend Prof. Stöckli for his permission to use this case of his.

There is also a rather pronounced oblique occlusal plane. The teeth are in good interdigitation, but the uppers as well as the lowers are slightly tilted to the hypoplastic right side. The upper second incisors are miss-

ing. The lower dental midline is also shifted to the right by half an incisor (Fig. 16g). Function, muscles and nerves are normal. The right masseter muscle is less voluminous than the left.

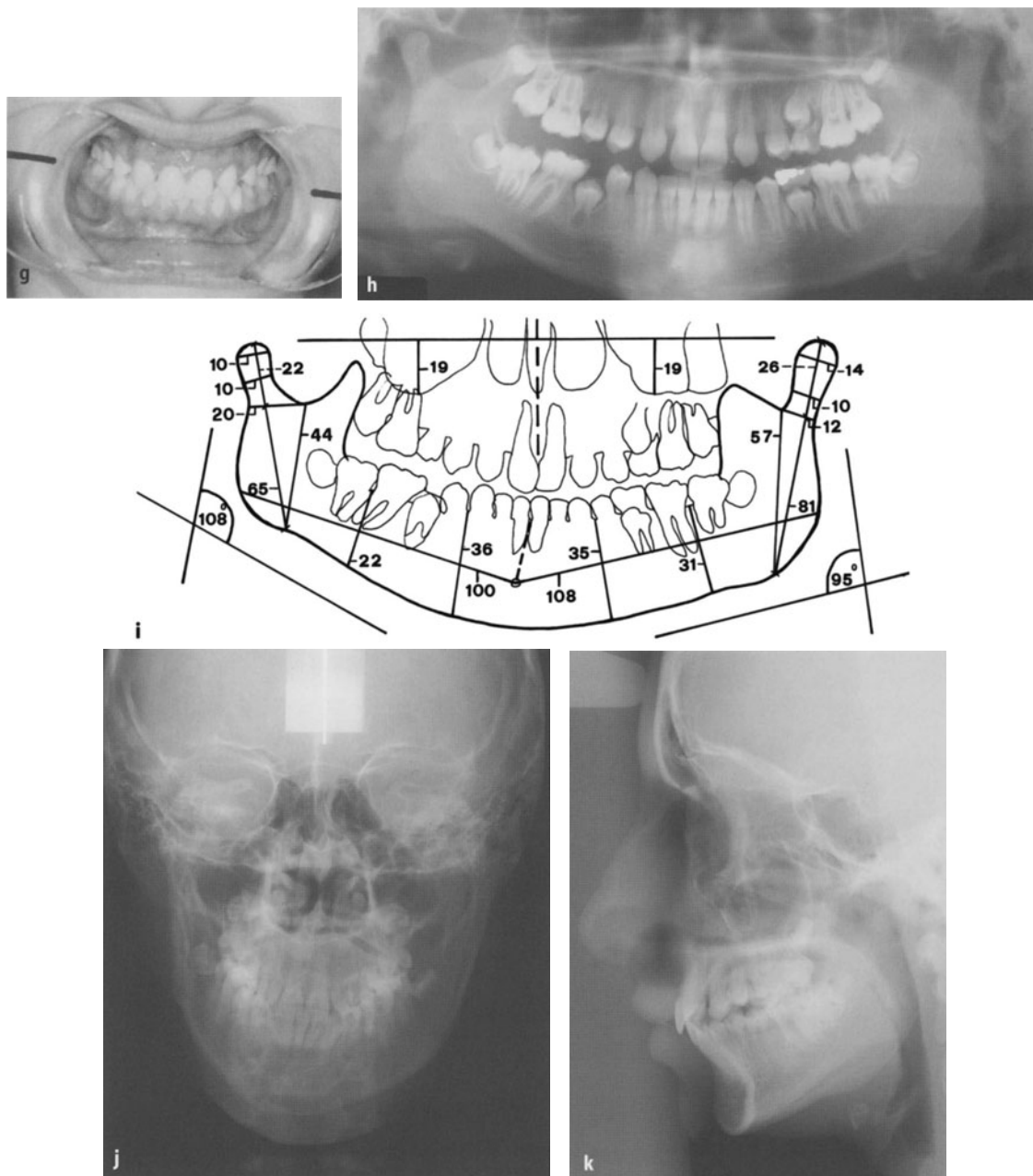


Fig. 16g–k. Case of unilateral congenital hypoplasia of the condyle. **g** The occlusion picture shows a shift of the mandible to the right side and an ascending occlusal plane towards the same side. **h** Panoramic radiograph of the mandible showing clear asymmetry between the right and left sides of the mandible with all the signs of a right side hypoplasia. **i** The measurement of the tracing of the panoramic radiograph gives clearer evidence of the degree of difference between left and right sides than expected by looking at the patient and her radiographs. **j, k** The p.a. projection cephalogram shows a more severe skeletal facial asymmetry than does the patient's front view (Fig. 16f). at the same age, while the lateral cephalogram discloses the vertical height discrepancy between the two bodies only

Radiographs. *Panoramic view* in a slightly open position at the age of 12 years and 5 months (Fig. 16h) shows that the two halves of the mandible differ noticeably in size and shape. There is no real shift of the midline of the lower teeth to either side compared with the upper. The slight mouth opening may have corrected a minor discrepancy between the upper and lower tooth midline. It is clearly noticeable that the smaller right condyle is further out of the glenoid fossa than the left. The structure and shape of the left hemimandible and its angle and of the condyle including its neck is almost that of a typical masseter hypertrophy or deep overbite, but also a bit similar to that of a H.H. The right mandible is smaller in the ascending ramus and body height. The angle is nearly normal with a slight, shallow antegonial notch and the condyle is small with fine trabeculations and no cortical covering. The right side of the maxilla has not grown down as much as the left side.

The measurement of the tracing (Fig. 16i) reveals a difference in length of the horizontal rami of 18 mm, of the ascending rami 7 mm, of the trunks 5 mm and of the articular processes 5 mm. In relation to these skeletal differences and in addition to the difference between the volume of the covering soft tissues of the two sides the patient's appearance only shows rather little asymmetry.

Cephalometric p.a. projection (Fig. 16j) shows a clear rotation of the lower half of the facial skeleton to the right side, with hypoplasia of the ascending ramus, deviation of the chin prominence to the right side, lack of maxillary height on the right side and reduction in size of the right maxillary sinus compared with that on the left side.

Lateral cephalometric view (Fig. 16k) shows a clear vertical discrepancy between left and right mandibular bodies and angle. Nothing else.

Mandibular p.a. projection in maximum mouth opening, TMJ-ramus tomograms, panoramic view in occlusion, scintigraphy of facial skeleton. All these requested investigations were not permitted by the parents.

Provisional Diagnosis. Congenital condylar hypoactivity of the right side with resultant hypoplasia of the hemimandible of the same side including the condyle; deviation of the prominence of the chin; shift of midline of the lower teeth to the hypoplastic side; hypoplasia of the masseter muscle of the same side.

Treatment Plan. Surgical correction of the asymmetry after growth has ceased, probably by rotation of the lower half of the facial skeleton including chin correction. In addition some contour filling of the hypoplastic right side may become necessary. I personally would use thin slices of subperiosteally-placed lyophilized cartilage. The zygoma may also need to be increased in prominence. It might have been wise to perform a high condylectomy very early, thus eliminating the negative growth

influence of the unilateral condylar hypoactivity and permitting the functional matrix to allow the affected mandible to develop normally as experienced in the case of Fig. 26. Then the maxilla could also have grown downwards to be equal to the other side. Another way of treatment would be a high condylectomy with simultaneous insertion of a costo-chondral graft, as early as possible. However, overgrowth to the other side would have to be anticipated.

Actual Treatment. So far none.

Discussion. There is no doubt about the existence of a hemimandibular asymmetry in this girl; however, it is not very severe in her facial appearance, but more apparent in the p.a. cephalogram. The question, whether it is due to a rudimentary form of H.H. of the left side, which might be suggested by the panoramic radiograph, or a hemimandibular hypoplasia of the right side is not really applicable since the whole asymmetry had already started in early childhood and it also includes the zygoma and the soft tissues on the same side. For a H.H. of the contralateral side starting that early, much more downward growth of the mandibular body would also have been expected. So, there remains only the question, is it a hemimandibular hypoplasia as a result of congenital or postnatally acquired condylar hypoactivity? The answer is again very clear. It has to be a congenital cause, because in the panoramic radiograph the condyle does not show any sign of secondary damage. As in congenital condylar hypoplasia the condyle always shows a very fine trabeculation and no sclerosis or surface irregularities, contrary to the picture after secondary damage to the condyle.

The fact that at the age of 1 year the mandibular asymmetry was noticeable, and yet it was not very severe at the age of 12 years and 5 months, supports the conclusion that the disturbance of growth in a congenital condylar hypoplasia may be much less severe than that seen when it has happened through postnatal infection damage. The type and amount of growth disturbance in this case is similar to a picture of mild damage acquired at the age of 5 years. It is interesting to assess that in spite of full function the affected hemimandible did not develop equal to the other side as has been experienced when iliac crest grafts are used to graft a ramus-condyle complex at a young age.

Conclusion. One might expect that congenital condylar hypoplasia and in infancy acquired secondary hypoplasia of the condyle might produce a similar clinical picture. This is not the case. Congenital condylar hypoplasia does not interfere that much with growth and development of the affected mandibular half and the maxilla as does condylar hypoplasia on the basis of an infection-caused damage in infancy.

The treatment might differ remarkably. In the case of damage due to a postnatal noxious event, one might decide on an early costo-chondral graft replacement of the damaged condyle or an early resection of the condyle only, in order to permit the functional matrix to allow the mandible to grow normally. In the case of a congenital hypoplasia, not as a part of a syndrome, growth may cease without severe asymmetry.

10.1.3 Unilateral Congenital Mandibular Hypoplasia in Cases of Hemifacial Microsomia (H.F.M)

In cases with hemifacial microsomia, there is not only a congenital hypoplasia of the condyle but also a unilateral embryonic developmental disturbance of a greater or lesser area in one half of the face, as a consequence of rupture of the stapodial artery in the 5–6th embryonic week. This was clearly demonstrated by Poswillo (1973) in animal experiments with suggestions for the reconstruction (Poswillo 1974). In these cases, the deficit of mandibular growth not only depends on the amount of damage to the condyle but also much more on the amount of destruction of the embryonic tissues of the whole region involved in the bleeding and diminution in blood supply from the ruptured artery. This involves the TMJ as well as the ramus and often the body of the mandible and surrounding soft tissues. In spite of the normal opening of the mouth existing since birth, the mandible of the involved side develops like an extreme form of unilateral condylar hypoplasia. As opposed to the cases of congenital hypo- or aplasia of the condyle, these cases lack the possibility of adequate growth stimulus by the so-called “functional matrix” on the involved side, as is the case in the normal development of the mandible. Hemifacial microsomia has no genetic background.

We have also observed cases with bilateral hemifacial microsomia. They were clearly asymmetrical (H. Obwegeser 1974, H. Obwegeser et al. 1985). The growth problem of the mandible and the surrounding tissue is the same as in unilateral cases. The final developmental result is not symmetrical in these cases. Each side depends on the site and extent of the damage caused by the bleeding.

Classification and Basics in Treatment Planning of Hemifacial Microsomia

Classification of a case of H.F.M. first requires a clear diagnosis of what is abnormal and to what degree. “*Pray every day to God that you do not become blind with open eyes*” (Principle Nr. 42). In general the affected area presents a 3-dimensional hypoplasia of a mild, medium or severe degree. All details must be recorded, specifically identifying separately bony and soft tissue abnormalities.

Several types of classification have been suggested. All of them try to classify separately the degrees of skeletal and soft tissue abnormalities. The most precise classification is the O.M.E.N.S.-classification, suggested by A. Vento et al. (1991). It classifies separately the orbit (O), the mandible (M), the ear (E), the facial nerves (N) and the soft tissue (S). Even the classification cannot indicate what kind of surgery will be required to achieve a good result. Each surgeon will have to give his personal answer to that question, depending on his diagnosis, vision and his surgical abilities. In order to achieve the best possible result in reconstructing the case, all abnormalities of each case have to be corrected. The plan for the reconstruction must adhere to the principle: “*In facial reconstruction, first the bone then the soft tissues*” (Principle Nr. 5).

Depending on the degree of the skeletal deformity almost every case requires an individual procedure for the correction of the skeletal abnormality (H. Obwegeser 1974; H. Obwegeser et al. 1985). After the age of 17 years “*Rotation of the lower half of the facial skeleton is the key to correction of facial height asymmetry*” (Principle Nr. 15) (H. Obwegeser 1970). With the insertion of a costo-chondral graft in infancy or early childhood, the main skeletal abnormality will be spontaneously corrected during growth in most cases (L. Kaban et al. 1988), but definitely not the lack of lateral growth of the base of the skull with the articular fossa. That area has to be built up surgically in order to place the reconstruction of the missing ramus in a lateral position equal to the final position on the normal side.

A general remark on the correction of hemifacial microsomia deformity seems necessary when I see the results of some, so-called very famous reconstructive surgeons: first of all facial incisions are contraindicated as nobody knows beforehand what kind of a scar the patient is going to produce. Although the use of an anastomosed iliac crest graft has a great advantage, but not at the price of an ugly scar as I have repeatedly seen. The same is true for the anastomosed free fat graft. This procedure not only includes the need for a large facial incision but also it cannot be certain that the fat graft will result in the accurate and ultimate correction. There can be too much or not enough volume, just as in the free non-anastomosed fat graft. But in the latter an additional amount of fat can always be added where it is needed, through a small, hidden incision. I hardly ever had need to add more fat. In some cases I had to excise the surplus as not as much resorption occurred as I had anticipated. I experienced complete resorption of the transplanted fat only twice. Both cases were lipodystrophy cases.

These remarks do not exclude that distraction osteogenesis has brought great benefit to these poor patients. But in spite of that, additional corrective surgery should not involve any incisions which will end with visible scars.

Hemifacial Microsomia After Growth Has Ceased (Fig. 17)

Sex and Age. Female, 21 years of age.

Aetiology and History. Congenital. The abnormality was fully recognizable at birth. The asymmetry became more obvious during growth. No relevant family history.

Clinical Findings. Very typical, rather severe degree of left side hemifacial microsomia (Fig. 17a–d) with severe hemifacial hypoplasia, deviation of the chin, oblique lip commissure and oblique occlusal plane with lack of vertical height and hypoplasia of the temporo-zygomatic area, with a very severe ear deformity and absence of an auditory meatus on the affected side. The oblique occlusal plane is severe and the mandible deviates to the left on opening. The facial structures on the left side are

rather short in the p.a. dimension and the soft tissues very thin due to lack of underlying musculature and subcutaneous tissue.

Radiographs. *Pantomogram; mandible p.a. in maximum opening; Water's view of the skull; p.a. and lateral cephalostat and others* (Fig. 17e–h). In all these radiographic projections the whole facial skeleton shows the typical anomaly of one of the many variations of hemifacial microsomia of a rather severe degree.

The supraorbital rims are at similar levels, but the left orbit is slightly deformed with its inferior rim lower than the right, due to the hypoplastic zygoma. There is severe



Fig. 17a–d. Case of unilateral severe hemifacial microsomia after growth has ceased. **a–d** Patient's presurgical appearance

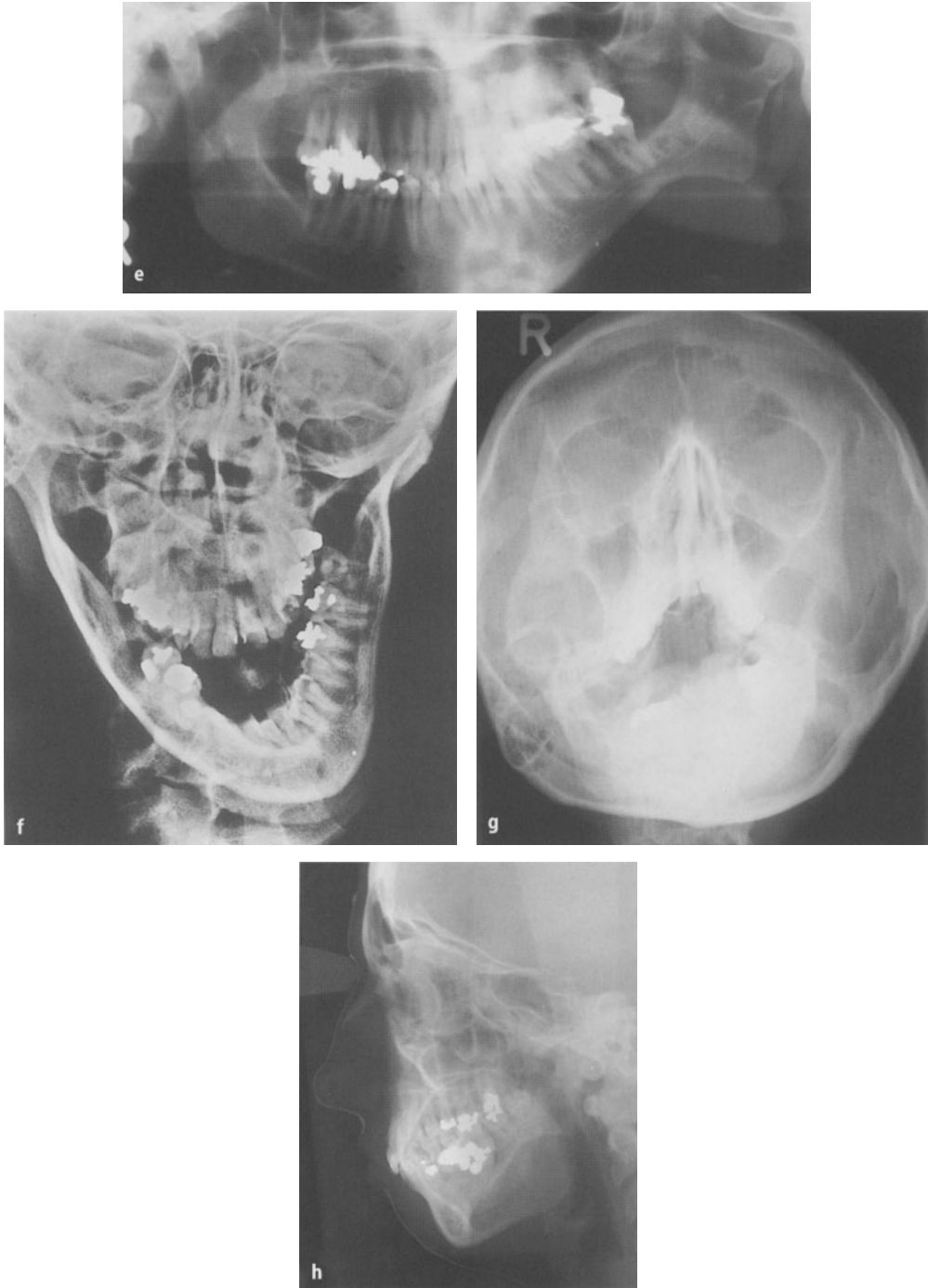


Fig. 17e–h. Case of unilateral severe hemifacial microsomia after growth has ceased. Presurgical **e** panoramic view, **f** mandible p.a.-projection in the open mouth position, **g** Water's view and **h** lateral cephalogram demonstrating the very severe skeletal hemifacial anomaly of the case, but with the supraorbital rims at the same level

shortness of the left side of the face, mainly due to maxillary and mandibular shortness in the vertical and horizontal dimension. The zygoma and in particular the temporal area is also hypoplastic on the left side. The right side shows absolutely normal skeletal structures.

As far as the mandible is concerned, all radiographs show the typical picture of very severe condylar hypoactivity, not resembling condylar aplasia or hypoplasia, but a shape similar to the skeletal picture seen after destructive osteomyelitis in early childhood, but with normal trabecular structure. There is very severe lack of vertical height of the ascending ramus as well as the horizontal ramus in its posterior half. The angle has a very short radius and is around 90° . In front of it is a very deep antegonial notch. The condyle is very small but within a normal form and shows normal, fine trabeculation.

Provisional Diagnosis. Hemifacial microsomia of severe degree on the left side.

Classification. According to the O.M.E.N.S.-classification: O2, M2b, E3, N0, S3

Treatment Planning Considerations. Although there are standard procedures for the correction of hemifacial microsomia, depending on the severity of the anomaly (H. Obwegeser 1970, 1974; H. Obwegeser et al. 1985), every single case of this congenital developmental disturbance has to be planned according to the structures involved and their associated deformities. In all these cases rotation of the lower half of the facial skeleton is necessary (Fig. 17i). As in this case a somewhat functioning TMJ existed, I decided to use this, in spite of its

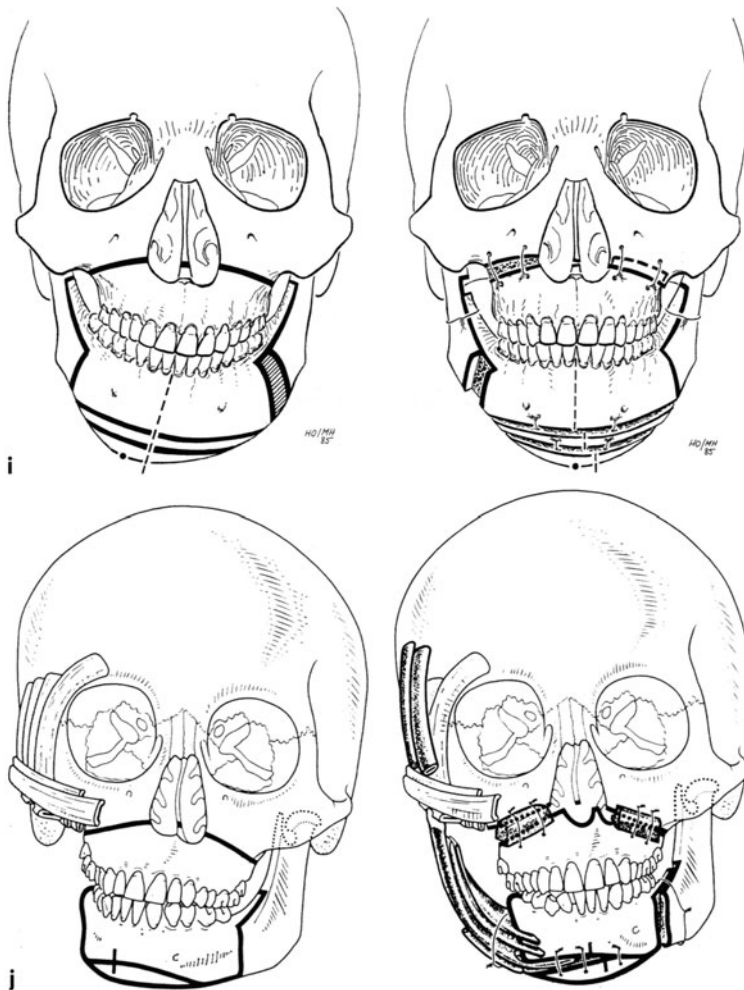


Fig. 17i-j. Case of unilateral severe hemifacial microsomia after growth has ceased. **i** Diagrammatic illustration of the author's first rotation of the lower half of the facial skeleton (from H. Obwegeser 1970). **j** Drawings of author's procedure applied for correction of very severe skeletal deformity in adult cases of hemifacial microsomia (H. Obwegeser 1974; H. Obwegeser et al. 1985b)

lack of normal forward movement, instead of building a new articular fossa, as I would do in cases where severe hypoplasia of the temporo-zygomatic area exists and the condyle, perhaps even a part of the mandible, is absent (Fig. 17j).

As it was necessary to increase the vertical height on the affected side by 19 mm, the condyle could be used only when the ascending ramus was elongated inferior to it. I planned to achieve this vertical increase in ramus length by a sagittal splitting procedure and adding a

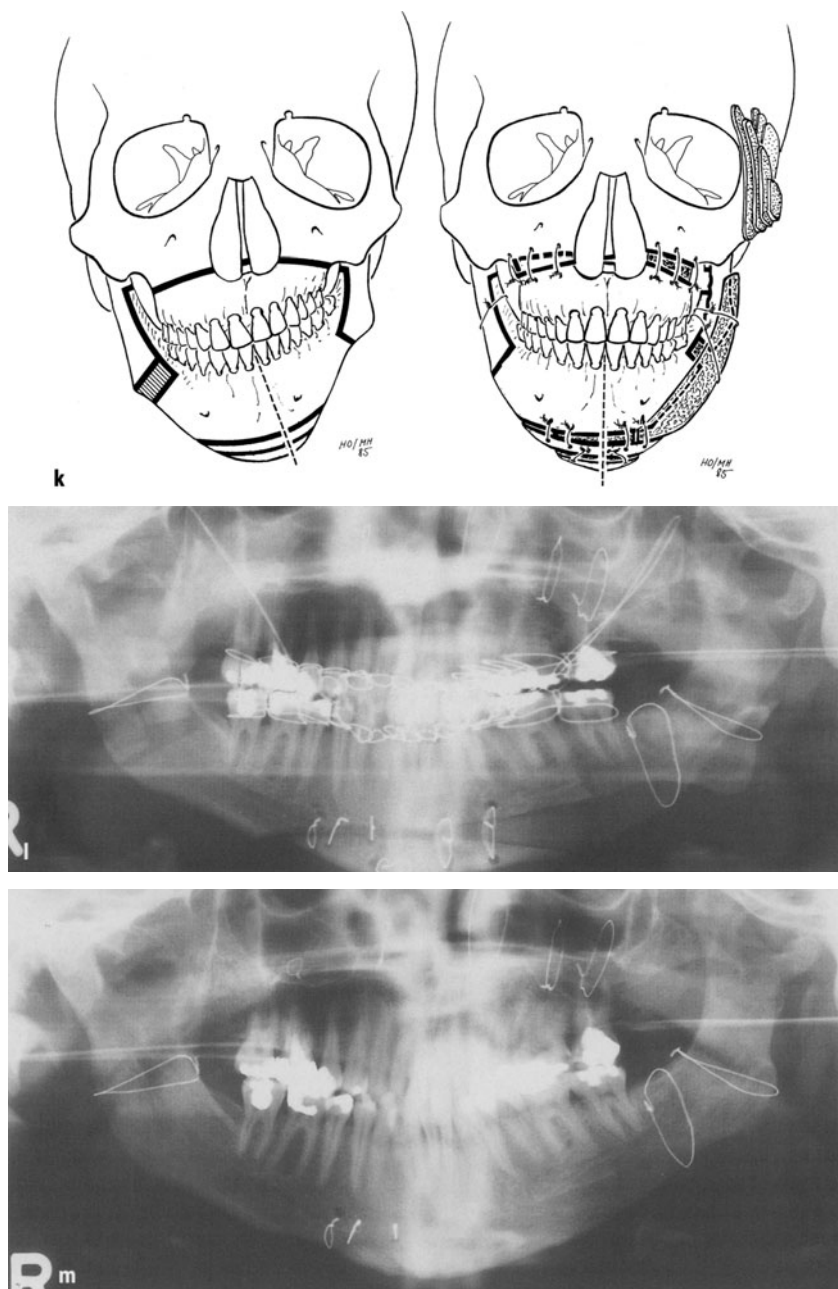


Fig. 17k-m. Case of unilateral severe hemifacial microsomia after growth has ceased. **k** Diagrammatic illustration of the procedure used in this case. Panoramic views of the mandible taken **l** 2 weeks and **m** 1 year and 5 months after the correction of the asymmetrical facial skeleton

large iliac crest graft on the lateral aspect of the split ramus for stabilization of the two fragments and to increase its thickness and contour. In accordance with this philosophy the following procedures were planned.

Treatment Plan. 1. In the first main intervention (Fig. 17k) vertical rotation of the lower half of the facial skeleton by a Le Fort I-type-osteotomy, raising the maxilla on the normal side by 4 mm and lowering it on the affected side by 15 mm and horizontal rotation to the non-affected side. An iliac cancellous bone graft to be

placed into the defect created in the left antral wall. Bilateral sagittal splitting of the ascending rami with rotation to the right, correction of the chin in two slices, fixation of a long iliac crest graft to the left horizontal and ascending ramus which will be placed between the symphysis of the mandible and the two chin slices. Build up the contour of the temporo-zygomatic area with subperiosteally onlayed lyophilized cartilage pieces and slices.

2. Slices of lyophilized cartilage to be inserted subperiosteally 6 months later, for further contour improvement of the left mandibular area.



Fig. 17n–q. Case of unilateral severe hemifacial microsomia after growth has ceased. **n** Mandible p.a. view 8 weeks, **o** Water's skull projection, **p** mandible p.a. projection and **q** lateral cephalogram 1 year and 5 months after correction of the skeletal asymmetry



Fig. 17r–z. Case of unilateral severe hemifacial microsomia after growth has ceased. Patient's appearance **r** with and **s** without occlusal spatula 18 months after the correction of the skeletal anomaly. **t, u** Patient 3 days after nonanastomosed free fat transplantation. The fat graft is suspended and held in place by subcutaneous sutures which are tied over shirt buttons. **v–z** The patient's final appearance 4 1/2 years after correction of the skeletal anomaly, i.e. 3 years after fat transplantation

3. Six months later, the lack of volume of soft tissues in the cheek area to be corrected by the implantation of a free non-anastomosed fat graft taken from the gluteal region.

Treatment Performed. At the age of 21–22 the correction was carried out to the forementioned plan, in three separate operations.

Postoperative Course and Result. Stage II of the treatment plan was carried out 5 months after the first operation. A fistula was present in the labio-mental fold. Under local anaesthesia, the previous operation site in the chin region was reopened from the vestibular sulcus. A piece of Supramid thread was found to be the cause of the skin fistula. A subperiosteal pocket was created along the left reconstructed mandible. Through that pocket, slices of lyophilized cartilage were onlayed, from the angle all the way forward to the chin.

Seventeen months after the first operation, the vertical height of both sides of the face was equal and the occlusal plane was horizontal. Out of the very distorted facial skeletal before surgery a normal, symmetrical skeleton was achieved, as a very important basis for the still necessary correction of lack of soft tissue volume (Fig. 17l–q). The occlusal plane was now horizontal (Fig. 17r). The left temporal area was still a little bulky. The two horizontal rami were equal in length, but the left mental protuberance was still less than that on the normal right side. The zygomatic area as well as the cheek including the horizontal ramus was still lacking in proper contour (Fig. 17s). The left nasal wing has moved remarkably inferiorly compared with the presurgical situation.

Stage III of the treatment planning was carried out 18 months after the first operation. All wires in the maxilla and mandible were removed. The chin prominence was rotated to the right side and simultaneously advanced on its left side. Lyo-cartilage block onlays were placed on the inferior border of the left horizontal ramus and on the left zygoma; at the same operating session a huge fat transplant, taken from the gluteal region, was fixed subcutaneously in the whole left cheek and even the mandibular, zygomatic arch and temporal areas. After undermining the entire area which had to be filled, the fat graft was contoured in accordance with the local deficiency requirements, however, with about 40% surplus in volume. It was held in place by percutaneous stitches which were fixed over shirt buttons. The threads had to pass through a few millimetres of subcutaneous tissue in the neighbourhood of the undermined area. For that task, mandibular and zygomatic awls were used. In this case an incision behind the ear was used to insert the fat into the large cheek cavity (Fig. 17t, u). Two weeks later the fat-suspending sutures were removed and a supporting bandage was applied for another week.

Three years after this last operation the patient's facial contour was very acceptable. Mandibular function

was normal. The left angle of the mouth was still further lateral than on the normal side. But altogether she was a good looking young lady without any visible facial scar. She covered her deformed ear with her hair. The correction of the skeletal as well as the soft tissue deformity remained stable (Fig. 17v–z).

The stability of the achieved correction of the severe skeletal anomaly is proven by comparison of the tracings of the lateral cephalograms of the presurgical situation and at 6 weeks and again at 1 year and 3 weeks after the correction (Fig. 17aa).

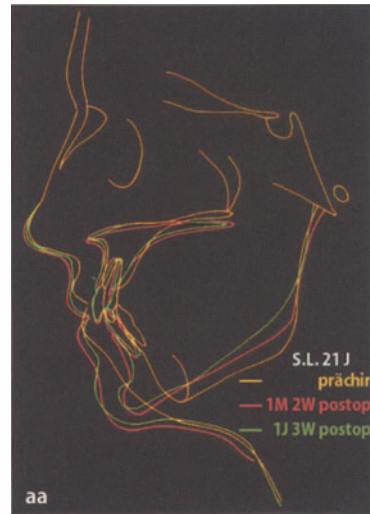


Fig. 17aa. Case of unilateral severe hemifacial microsomia after growth has ceased. **aa** Tracing of the lateral view of the facial skeleton before (yellow), 6 weeks (red) and 1 year and 3 weeks (green) after correction of the facial asymmetry

Histology. The rudimentary condyle was left in situ and not removed for histological examination.

Discussion. Although hemifacial microsomia presents as congenital hypoplasia of the condyle it is, in its consequences, entirely different to cases of isolated congenital aplasia or hypoplasia of the condyle. The lack of normal skeletal growth and development in these cases is probably caused only partially by the developmentally damaged condyle. It is mainly due to embryonic damage to all the tissues in the affected areas due to rupture and bleeding of the stapedia artery with consequent lack of blood supply to the embryonic tissues in the involved area. While in congenital aplasia or hypoplasia of the condyle the condylar hypoactivity affects mainly the height of the ascending ramus, the embryonic bleeding interferes with the development of the mandible very similarly to the osteomyelitic destruction of the mandible in early childhood. However, osteomyelitis does not produce a developmental ear deformity and absence of an auditory meatus and does not influence so

severely the musculature and subcutaneous soft tissues as does hemifacial microsomia. While in arthritis or osteomyelitis cases the early removal of the damaged condyle might give the mandible a chance to develop normally, I doubt whether this would also be the case in hemifacial microsomia.

Conclusion. Unilateral and bilateral hemifacial microsomia may present hemifacial asymmetry in any de-

gree, from very slight to very severe. It is, however, not a true hemimandibular hypoplasia only, as in those cases where the condyle alone has been damaged. The hemimandibular hypoplasia in these cases is not due to condylar hypoactivity alone. The diagnosis is easy and the corrective surgery has to be planned according to the extent of the bony and soft tissue defects. Facial incisions for reconstructive surgery of the facial skeleton can and must be avoided.

Unilateral Hemifacial Microsomia in Childhood (Fig. 18)

Sex and Age. Female, 6 years 11 months.

Aetiology and History. Congenital; parents came with the question "when and what kind of treatment should be done?" No relevant family history.

Clinical Findings. Appearance of typical hemifacial microsomia in childhood of medium severity degree on the right side (Fig. 18a–f). In closed and open mouth positions, severe asymmetry of the face with deviation of the chin to the right, with oblique lip commissure and oblique occlusal plane; the right corner of the mouth is further posterior and higher than on the left; the right external ear is deformed with skin tags attached to the ear lobe. No acoustic meatus is present. Flatness of the right zygoma and cheek area. Severe crossbite on the right side. Full complement of deciduous teeth with eruption of permanent lower anteriors. Patient otherwise absolutely normal.

Radiographs. *Panoramic view* (Fig. 18g) and *p.a. mandible* in maximal opening, and *Water's view*: On the right side there is no condyle, no glenoid fossa, no ramus, no angle and the mandibular body is missing as far forwards as the canine region. The alveolar process still contains the deciduous molars without a mandibular base under it. On the left side, the mandible is absolutely normal in shape and structure but severely deviated to the right side. The Water's view of the middle third of the facial skeleton shows a very hypoplastic zygoma and zygomatic arch on the right side and a much smaller maxillary sinus than on the left side. The right infraorbital rim is slightly lower than that on the left.

Provisional Diagnosis. Typical hemifacial microsomia of severe degree on the right side with severe skeletal defects and deformity of the affected side. Classification according to the O.M.E.N.S. type: O₃, M₃, E₂, N₀, S₃.

Treatment Plan. 1. Reconstruction of the hypoplastic zygomatic arch and missing glenoid fossa with split rib grafts and cartilage layers (see Fig. 19c, d) and simulta-

neous reconstruction of the missing parts of the mandible with a partially decorticated costo-chondral graft shifting the mandible into the midline.

2. Wait until symmetry of the mandible is achieved through the growth of the costo-chondral graft and then further corrections as required. Correction should be completed by the age of 18.

Actual Treatment. The first step in the treatment plan was carried out at the age of 6 years 11 months, according to the plan. Two 10 cm long costo-chondral grafts were removed from the right side. They were partially decorticated and one was used for the reconstruction of the hypoplastic zygomatic arch and missing glenoid fossa, and the other for the reconstruction of the missing mandible as far forward as the chin which was fixed with wires into a groove prepared on the lateral aspect of the chin. Where the glenoid fossa and the articular eminence would normally be found, there was nothing but a plain bony area. A temporal approach was used for the reconstruction of the zygomatic arch and the glenoid fossa (H. Obwegeser et al. 1985) and the reconstruction of the missing mandible was performed via the oral route. Following the fixation of the rib graft onto the existing chin area, the mandible was immobilized with intermaxillary fixation for 5 1/2 weeks (Fig. 18h–j). At the end of the reconstruction of the skeletal defects the skin tags in the ear region were excised. Post surgery there were no problems or complications.

Further Course. One year after the primary reconstruction there was still marked asymmetry of the face and a very oblique occlusal plane (Fig. 18k, l). At age 10, 3 years after the primary reconstruction, the right side of the mandible had grown downwards and shifted the chin further towards the midline (Fig. 18m, n). Mouth opening was fairly good, but still with some deviation to the right side. When the teeth were in occlusion only a small shift of the midline of the lower teeth to the right remained.

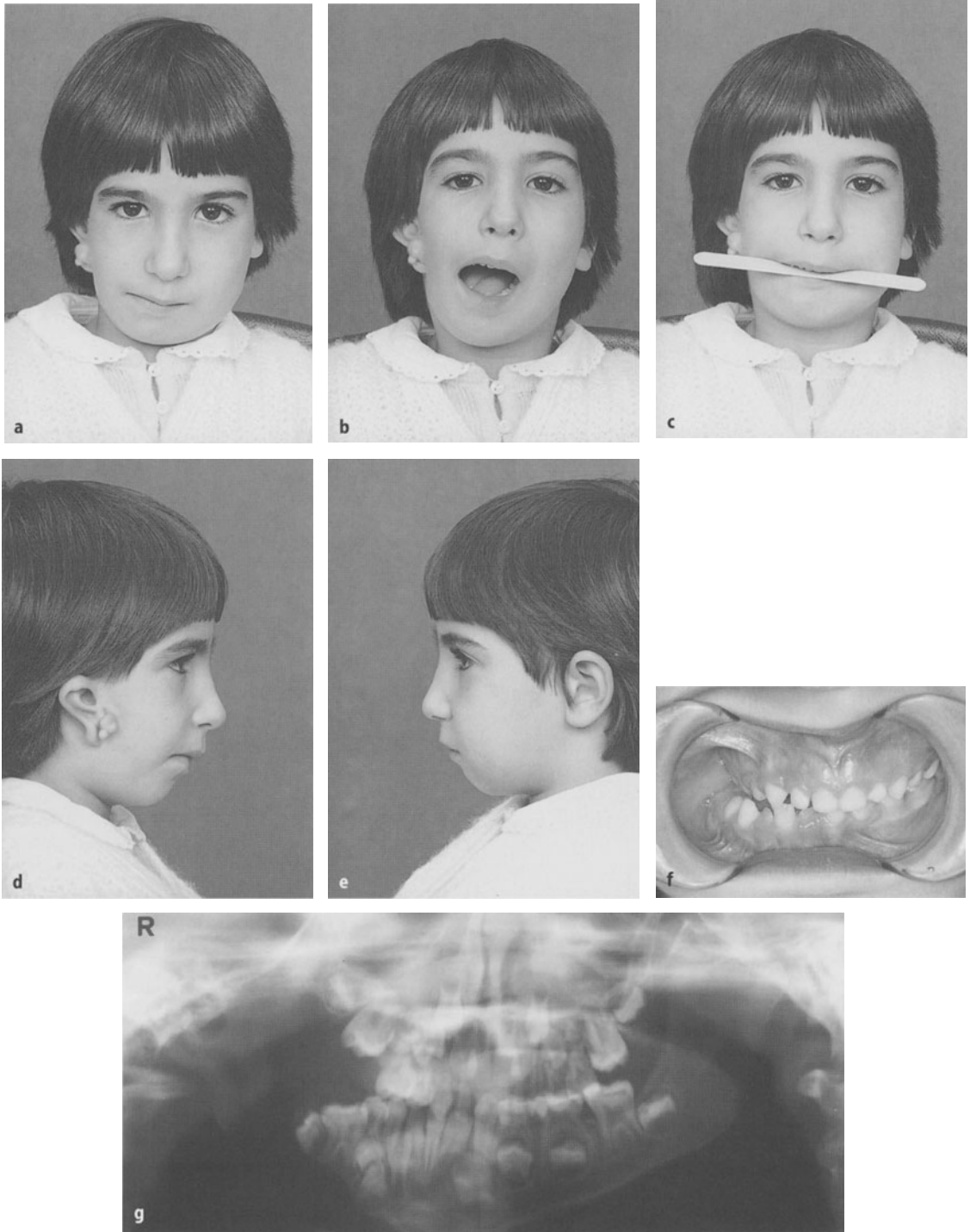
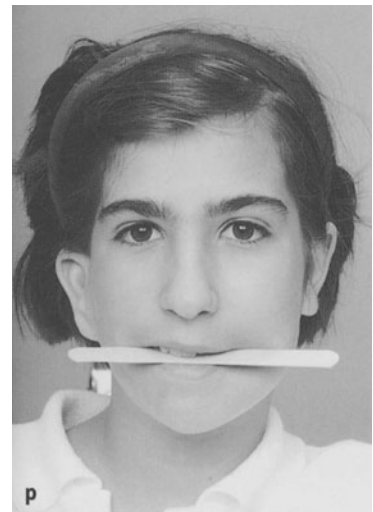
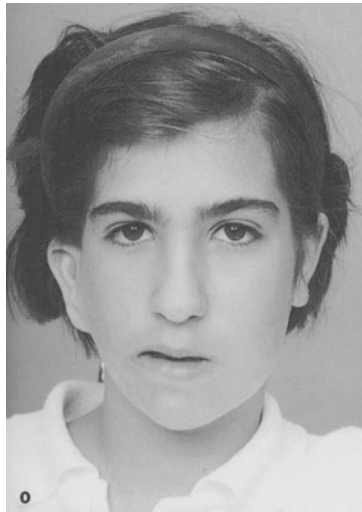
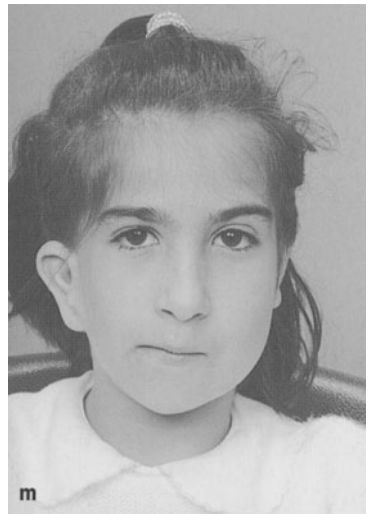
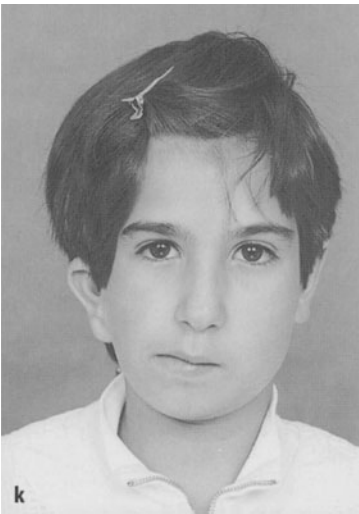
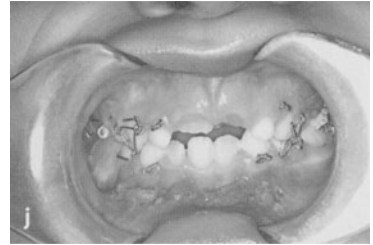


Fig. 18a–g. Unilateral hemifacial microsomia with a very severe skeletal defect at the age of 6½ years. **a–f** Patient's appearance and occlusion before surgery. **g** Panoramic view disclosing the skeletal background of the deformity: absence of right ascending and main part of horizontal ramus, severe hypoplasia of the zygomatic complex including absence of a glenoid fossa

Fig. 18h–p



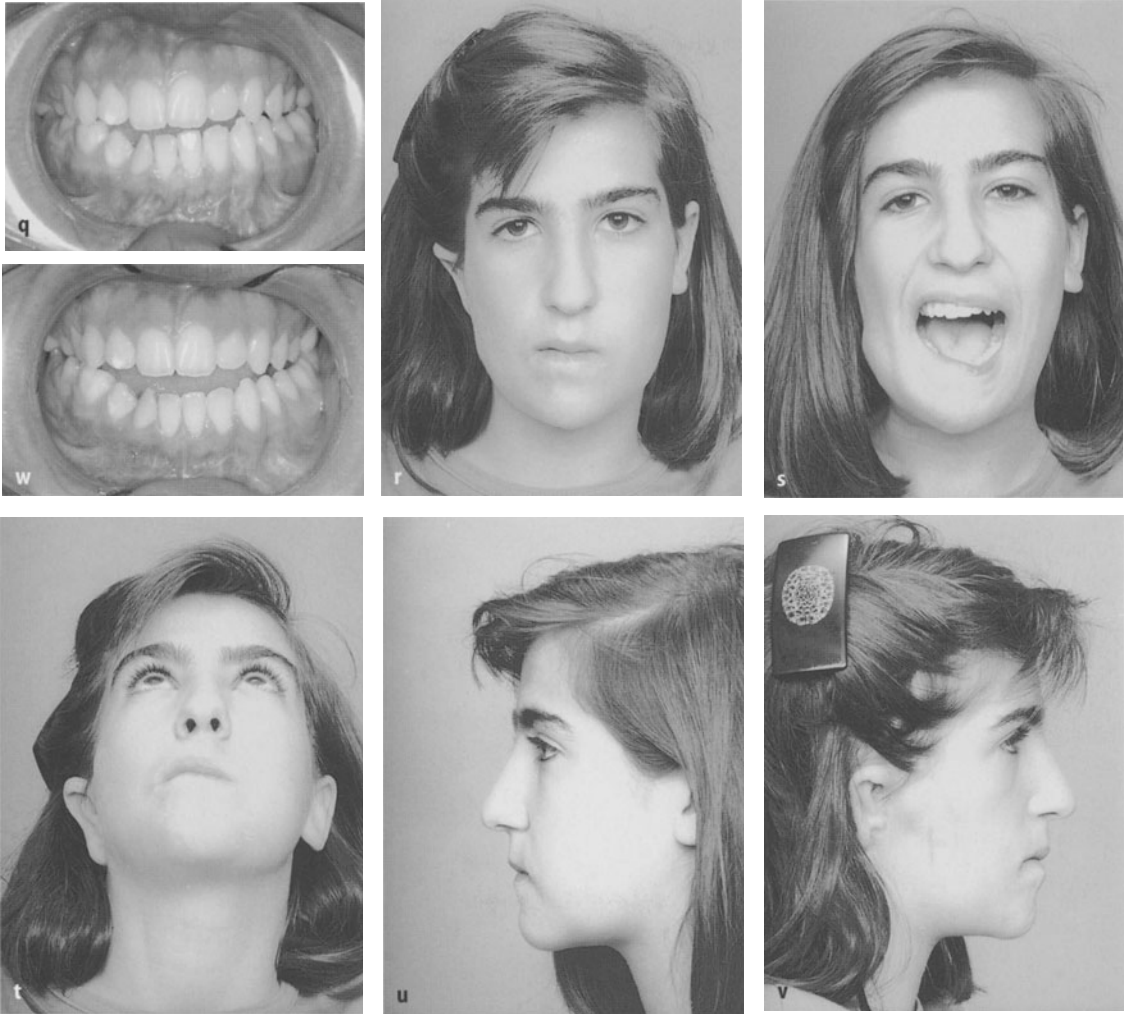


Fig. 18h-w. Unilateral hemifacial microsomia with a very severe skeletal defect at the age of 6 $\frac{1}{2}$ years. Patient's **h** front view and **i** from below, 5 $\frac{1}{2}$ weeks after reconstruction of the hypoplastic zygomatic arch and missing glenoid fossa and simultaneous reconstruction of the missing parts of the mandible. **j** Achieved occlusion, with intermaxillary fixation. **k, l** Patient's appearance and occlusal plane situation 1 year after surgery. The difference between left and right facial heights has increased. **m, n** Patient's appearance and mouth opening 3 years after surgery. **o-q** Patient's front view, occlusal plane and occlusion at age 12 $\frac{1}{2}$ years (6 years after surgery). The occlusion is shifting forwards and to the left. The occlusal plane is now almost horizontal. **r-w** Patient at 15 years and 1 month with almost symmetrical facial contours after some additional cartilage onlays and correction of chin asymmetry, but now with deviation of the occlusion to the normal side by the width of one lower incisor and with an open bite. In the profile views both horizontal rami seem of equal length

My successor, H. Sailer, then took over the case. At the age of 12 years, 5 years after surgery, the occlusion was already almost normal with no deviation of the midline of the lower teeth. At the age of 13, 6 years after surgery, there was already nearly complete facial symmetry (Fig. 18o–q). The occlusal plane was almost horizontal. The midline of the lower teeth had shifted over to the left side. One year later Prof. Sailer made some improvement in the contour by subperiosteal implantation of slices of lyophilized human cartilage and correction of the chin asymmetry. At the age of 15 years and 1 month, the outward appearance was very good, almost symmetrical, however with a severe anterior open bite and a shift of the occlusion to the non-affected left side. The mouth opening was fairly good with almost no deviation (Fig. 18r–w). Further corrections will not be difficult and should be routine. Of the rather radical decortication of the rib graft and its superficial perforations, when the rib was implanted, nothing visible of that preparation remained 8 years later (Fig. 18x, y). The costo-chondral graft has not only grown in length but has also become so thick that a sagittal splitting procedure could now be performed whenever necessary. It is interesting to note that underneath the cheek teeth on the

operated side an almost normal mandibular body has developed out of the rib.

Discussion. In cases of hemifacial microsomia there is no natural growth potential which could correct the facial skeletal deformity. In early childhood, there is an absolute contraindication to rotation of the lower half of the facial skeleton as suggested for patients at the end of the growth period. However, for psychological reasons as well as for skeletal developmental and for occlusal reasons, one should reconstruct the missing skeletal parts very early. If there is severe hypoplasia of the temporo-zygomatic area and no glenoid fossa exists in an acceptable position, the temporo-zygomatic complex can be built up very early in life using split rib grafts with cartilage for the glenoid fossa. As there is normally not enough cartilage or ribs available for building up the temporal contour by the necessary amount, slices of lyophilized cartilage, covered laterally by a micropore filter, are very useful for that purpose.

For the reconstruction of the missing ramus in children, I like superficially decorticated, full rib grafts with attached costal cartilage, hopefully obtaining further growth of the reconstructed missing mandible. A 10 cm

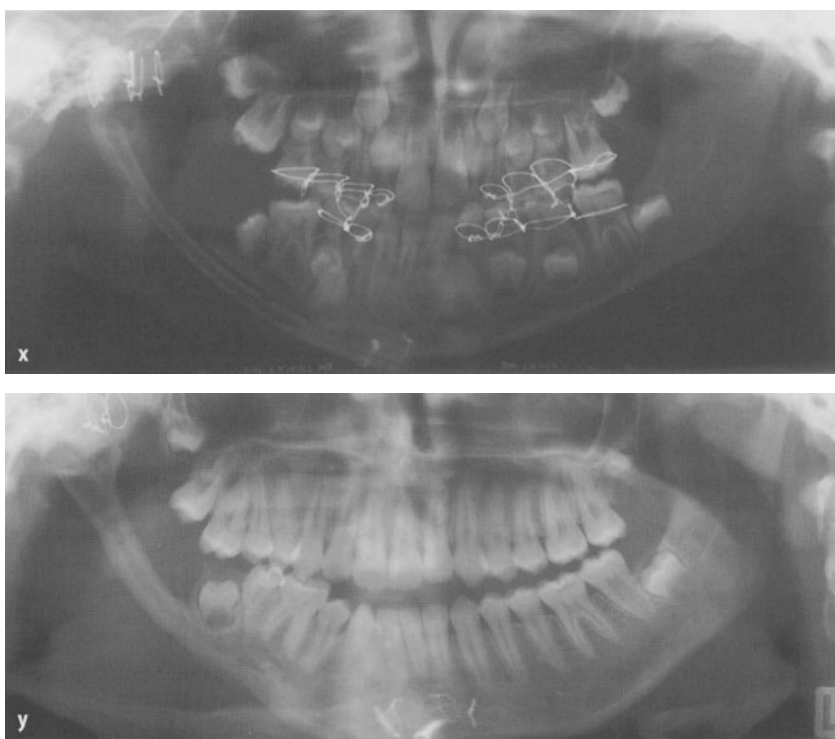


Fig. 18x, y. Unilateral hemifacial microsomia with a very severe skeletal defect at the age of 6½ years. **x** Panoramic view 2 weeks after reconstruction of the zygomatic region with a glenoid fossa and of the mandibular defect by a costo-chondral graft. It shows well the partial decortication of the rib graft with some superficial perforations through the thin cortical plates. **y** The reconstructed mandible at the age of 15 years and 1 month (almost 8 years after the reconstruction). The rib graft has not only gained remarkably in length but also in thickness. It can now easily be utilized for a sagittal splitting procedure

long rib graft will become necrotic in its middle section if no steps are taken to ensure its marrow makes connection with the surrounding soft tissue. For that purpose the rather thick cortical tube is superficially decorticated with some additional spots of complete decortication (see Fig. 18x). *“A graft covered by thick cortical bone is good for stability but has a poor survival rate”* (Principle Nr. 58). The graft will grow forwards and downwards and will also permit the maxilla to grow downwards and laterally, thus correcting the oblique occlusal plane and possibly overcorrecting the asymme-

try. The final correction will then be much easier to perform after growth has ceased.

Conclusion. In cases of hemifacial microsomia we expect lack of growth of the mandible depending on the amount of embryonic damage to the area. However, with early reconstruction of the missing glenoid fossa and ascending ramus with rib and costo-chondral grafting, growth can be expected to occur and make the later reconstruction of any remaining deformity much easier.

Hemifacial Microsomia in Infancy (Fig. 19)

Sex and Age. Female, 1 1/2 years of age.

Aetiology and History. Congenital. Parents wanted to know what should be done, and when. No relevant family history.

Clinical Findings. Rather pronounced hemifacial hypoplasia on the left side. The left ear is almost normal, but protrudes like a bat ear. Hearing on that side is zero. The chin is far over to the left side. There is moderate macrostomia on the same side and also hypoplasia of the temporo-zygomatic area. The deciduous teeth, as far as erupted, are in perfect occlusion, however, in a rather oblique plane, as is the labial commissure (Fig. 19a–c).

Radiographs. *Panoramic view only* (Fig. 19d), in view of the age of the patient. In the panoramic X-ray, the right

half of the mandible appears absolutely normal in shape and structure. The left half shows a flat concavity at the lower border of the body, but a normal angle and structure of the ascending ramus. The ascending ramus is shorter than that on the opposite side. The condylar neck is also shorter and the head is smaller and almost translucent. There is a pronounced oblique occlusal plane and shortness in vertical height of the left side of the face. The chin is shifted to the left and so is the midline of the lower teeth in the slightly open position of the mandible. While on the right side there is a normal auditory meatus this is not true for the left side where the meatus is almost unrecognizable. There was no hearing on the left side.

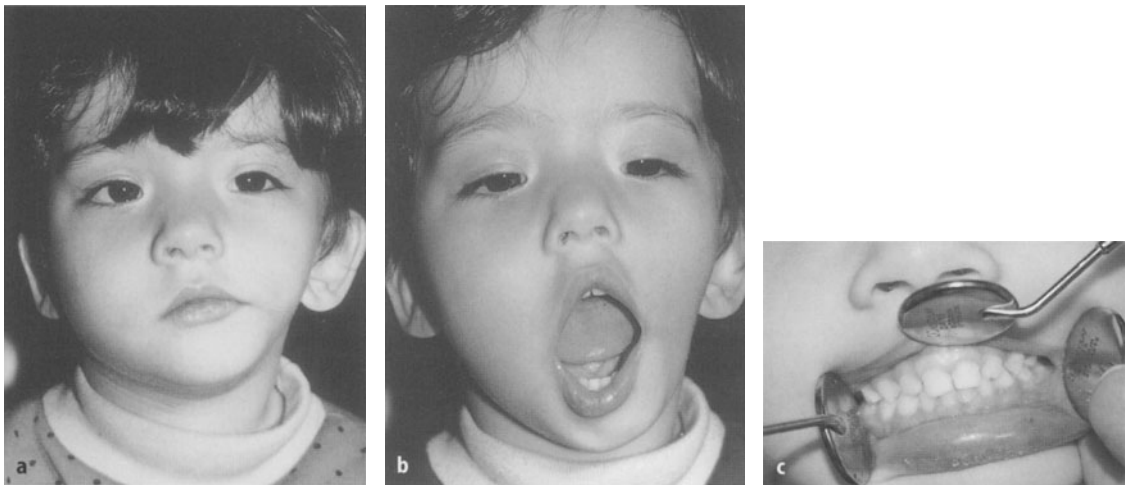


Fig. 19a–c. Hemifacial microsomia in infancy. **a–c** Front view in closed and open mouth position and occlusion of the 18-month-old girl with hemifacial hypoplasia due to a hemifacial microsomia

Provisional Diagnosis. Hemifacial microsomia with pronounced degree of hemifacial hypoplasia and deviation of the chin, a moderate macrostomia and an almost normal external ear.

Treatment Plan. In order to permit lengthening of the left ascending ramus, the hypoplastic condyle should be removed and a costo-chondral graft be inserted as an onlay graft and placed into a newly created glenoid fossa.

Actual Treatment. At the age of 1½ years, the temporal approach via a retro-tragal temporal incision (H. Obwegeser 1985) was used to perform the necessary work in the TMJ region. The very hypoplastic condyle was resected (Fig. 19g). I do believe that in cases with a hypoplastic condyle, normal mandibular development cannot be expected. Via an oral approach, a superficial decortication of the ramus area was performed in the form of a shallow groove where the rib graft was to be at-

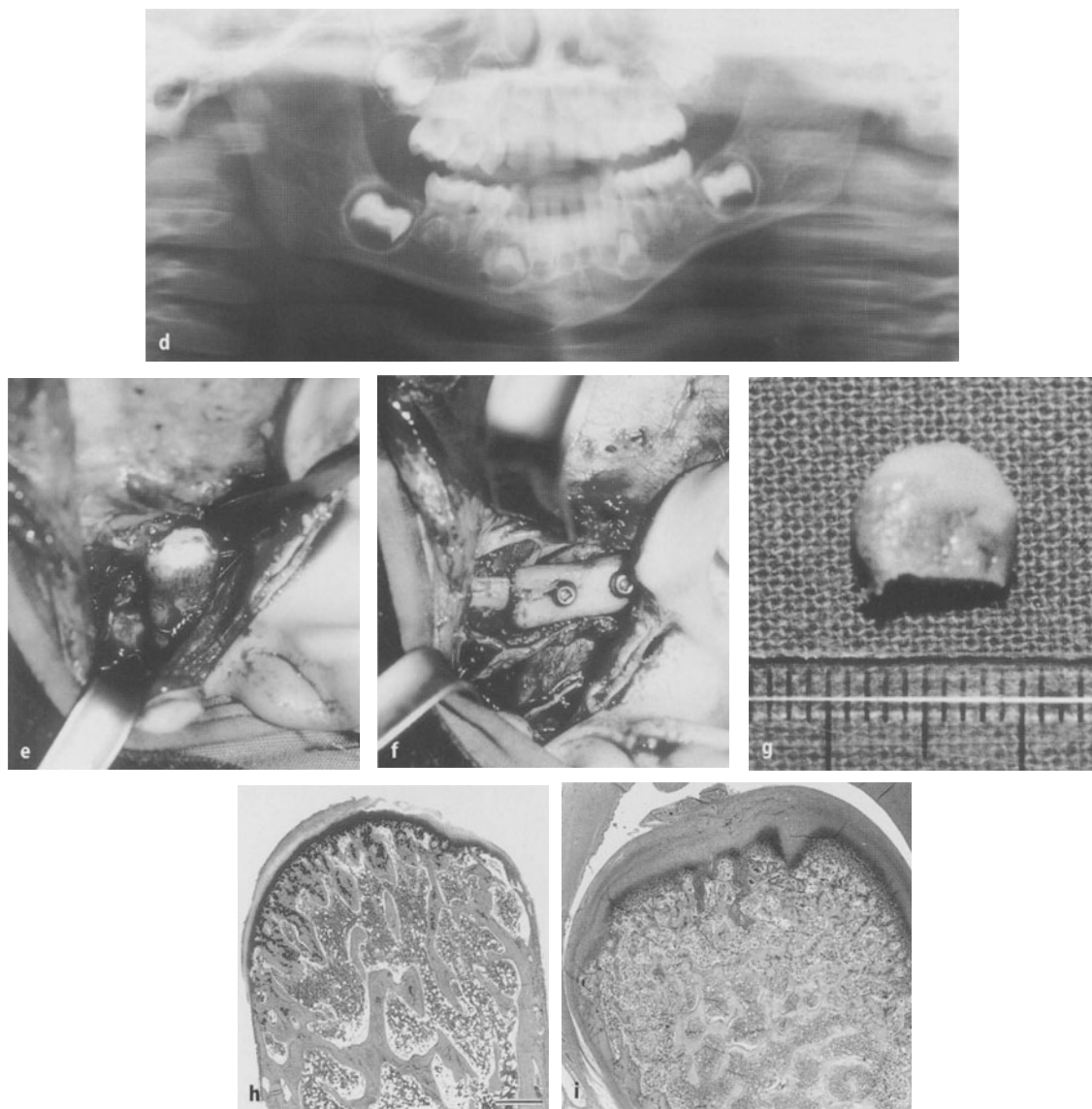


Fig. 19d-i. Hemifacial microsomia in infancy. **d** The panoramic view does not show that severe a defect of the mandible on the affected side as one would expect according to the patient's appearance. **e** The costo-chondral graft is placed lateral to the original TMJ area. **f** Above the rib graft the temporal region is built up with pieces of a rib with a glenoid fossa underneath. **g** The resected condyle of the hypoplastic ramus. **h-k** Histological comparison of the resected condyle (**h,j**) and a normal specimen from a 15-month-old boy (**i,k**). Overviews (**h,i**) show the reduced overall size of the affected specimen (**h**) which, probably as a result of the resection, lacked part of the superficial articular layer and the natural joint surface

tached (C. Lindquist et al. 1986). Then the initially taken and partially decorticated costo-chondral graft was fixed to it with two circumferential wires. They were preferred to screws as screws might easily split the thin rib and could also interfere with the mandibular nerve. As the rib graft was attached laterally to the ascending ramus, it will not only increase the contour of the ascending ramus region, but it will also end laterally in the temporal region (Fig. 19e). The hypoplastic temporal region was built up and the new glenoid fossa reconstructed. Two small pieces of rib were fixed with two screws to the temporal bone (Fig. 19f). Two slices of cartilage were placed underneath to form a glenoid fossa. The graft with its chondral end rested on them. The extra- and intraoral wounds were closed without any drainage. The day after surgery the child was able to open her mouth without difficulty. It is to be expected that the costo-chondral graft will produce enough length for the ascending ramus to become normal and will shift the chin over the midline as observed in other cases and will also permit downward growth of the maxilla. I have operated upon that charming child recently, in a foreign country, as a guest surgeon; unfortunately I will not be able to follow the late result in this case.

Histology. The resected hypoplastic condyle was of special interest for histological examination. It was interesting to find out if there was any and what type of histological difference it might show. Compared with those of a normal condyle of the same age (Fig. 19i,k), the ex-

isting layers of connective tissue and cartilage were histologically normal (Fig. 19j). There was a continuous proliferative layer of loose fibrous connective tissue and a subjacent layer of hypertrophic growth cartilage. Also, endochondral ossification of this growth cartilage seemed to have been functioning normally, as numerous chondroclasts were present in the subchondral spaces, while osteoblasts covered newly formed bone trabeculae.

The age-related size of the removed condyle, however, was clearly abnormal (Fig. 19h): the antero-posterior diameter of the specimen corresponded approximately to that seen in a normal new-born, and the hypertrophic cartilage was much too thin, its absolute thickness corresponding approximately to that found in a normal 2.5-year-old child. Even when related to the reduced overall size of the affected condyle, the thickness of the hypertrophic layer was abnormally small. Although this is not conclusive proof, it still suggests that growth of the condyle had been less than normal.

Discussion. The hypoplastic condyle was removed and a costo-chondral graft was inserted as an onlay graft and placed into a newly built glenoid fossa, in a 1½-year-old child with an unilateral medium degree of a hemifacial microsomia. Within a few years the mandible should, according to experience, grow downwards and forwards thus correcting the mandibular and chin asymmetry and permitting the maxilla with its teeth to grow downwards also, as also experienced by L. Kaban et al. (1988).

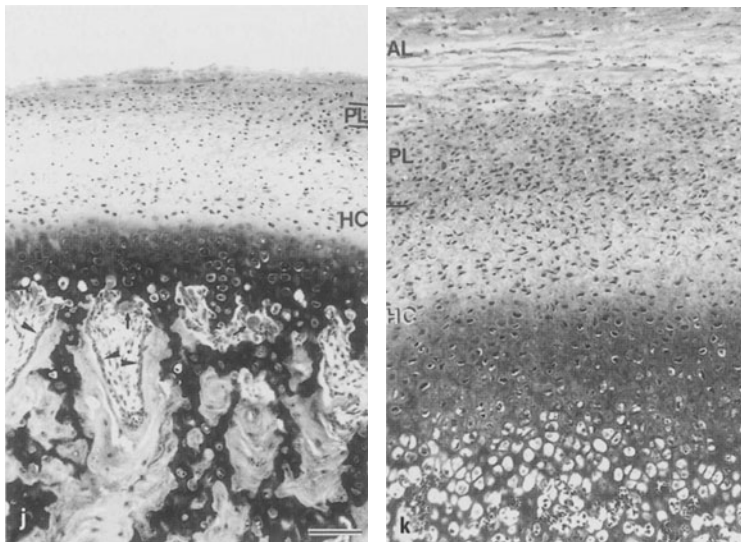


Fig. 19j, k. Hemifacial microsomia in infancy. For this reason, details from the articular tissues (j,k) are aligned at the border between the articular (AL) and proliferative (PL) layers. Note the reduced thickness of all tissues, including the hypertrophic cartilage (HC), of the affected condyle (j), although the presence of chondroclasts (arrows) and seams of osteoblasts (arrowheads) suggests ongoing endochondral ossification. Plastic sections, toluidine blue; original magnifications: h,i $\times 10$ (bar in h=1 mm); j,k $\times 115$ (bar in j=100 μm)

Conclusion. A hypoplastic condyle, independent of its aetiology, should be removed at any stage of the growth period in order to eliminate its negative growth influence. Depending on the aetiology, a costo-chondral graft has to be inserted to permit normal or even over-

growth of the hypoplastic mandible. The histology of the condyle showed normal structural layers but with much too thin a chondral layer compared with a normal case of the same age.

10.1.4 Hemimandibular Hypoplasia Due to a Perimandibular Cavernous Haemangioma (Fig. 20)

Sex and Age. Male, aged 17 years and 2 months.

Aetiology and History. Slight facial asymmetry became noticeable during puberty. On account of occlusal problems, the patient was referred to the Department of Orthodontics at the Zürich Dental School. Because of the abnormal radiological findings he was referred for surgical correction of the retromandibulism and the oblique occlusal plane. The question arose whether or not hemifacial microsomia was the cause of the asymmetry. No relevant family history.

Clinical Findings. The patient presented a rather complex atypical unilateral facial asymmetry. He was referred with the diagnosis hemifacial microsomia (Fig. 20a–d). Looking at him from in front there is a slight left-sided hemifacial hypoplasia noticeable, with some flatness in the periorbital-zygomatic and preauricular regions on that side. In addition there is retromandibulism with a slight shift to the left side. The amount of mouth opening is not restricted, but with deviation to the left. There is quite an oblique occlusal plane, ascending to the left side and the left corner of

the lip is more lateral and higher than that on the right. There is a full dentition with the exception of a missing right lower first bicuspid. The occlusion is that of a severe class II with deviation to the left side (Fig. 20e). The left ear is a bat ear type. It inserts further inferiorly than does the one on the right. On both sides there is normal hearing.

Radiographs. The panoramic view shows a very abnormal left mandible. The posterior part of the body is much less high than normal. There is no angle, but instead, a concavity with some bony spicules. The posterior half of the ascending ramus is transformed into a long condylar neck with a finger-like shape. There is no real condyle on its upper end. The second and third molars are impacted and the coronoid process is also rather long and fingerlike (Fig. 20f). The left maxilla seems slightly compressed and its base is higher and shorter than that on the right side. The right mandible looks normal. The left mandible is not only deformed, but its ascending ramus has also developed in an abnormal lat-

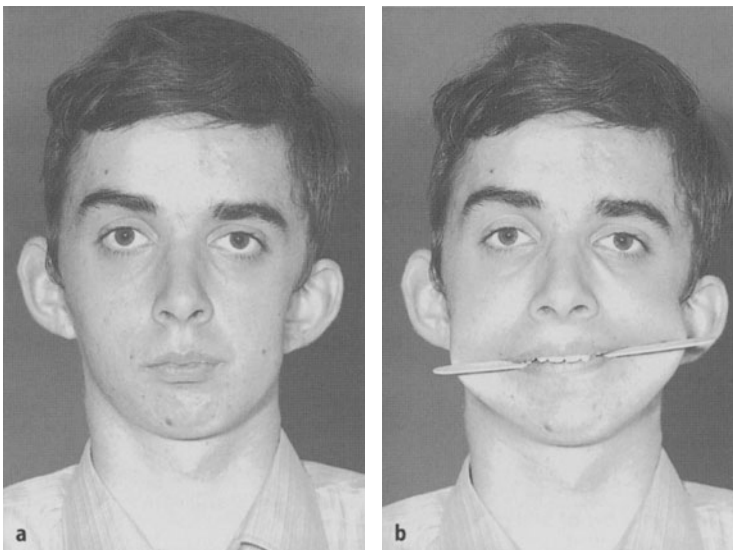


Fig. 20a, b

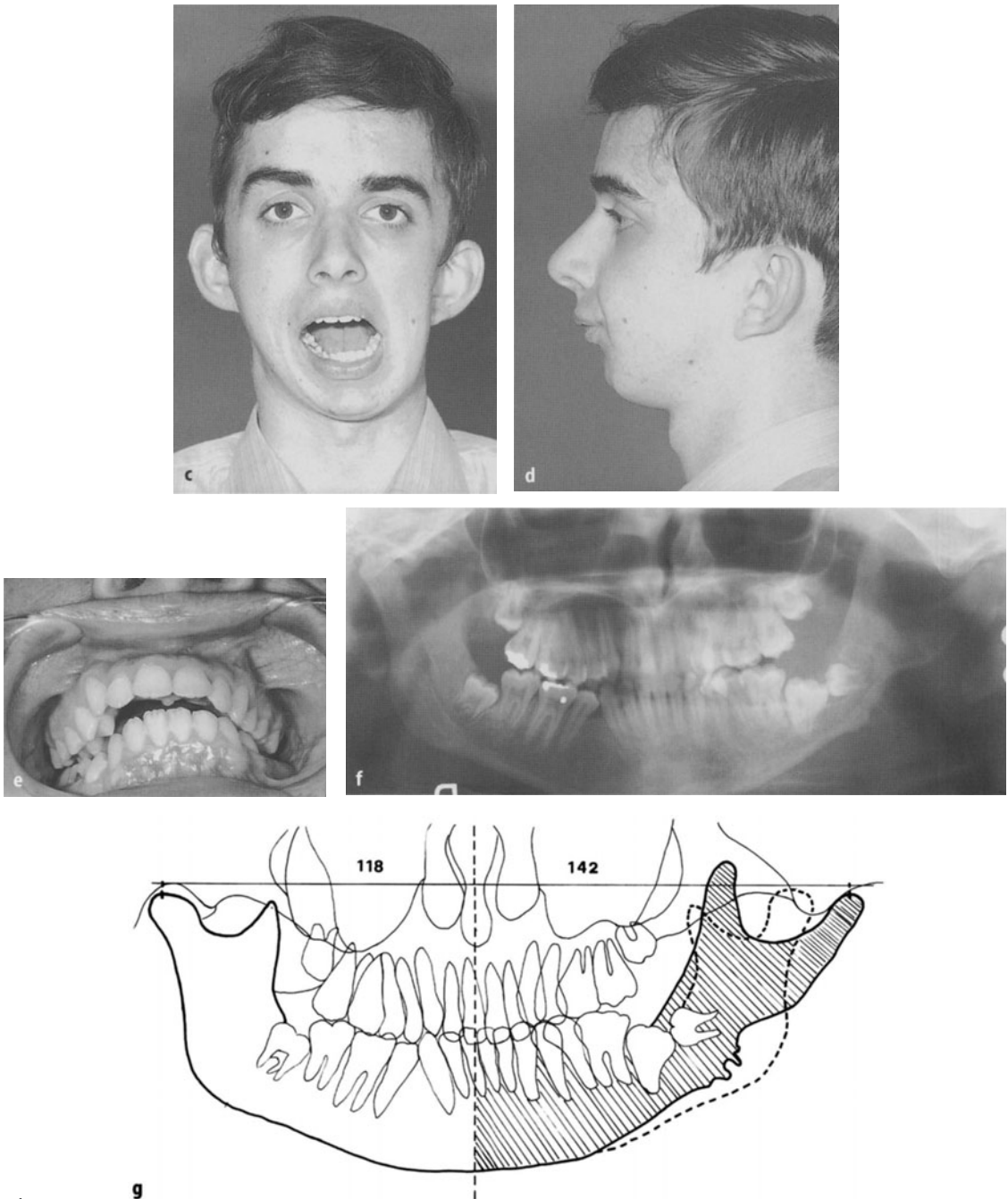


Fig. 20a–g. Hemimandibular and hemimaxillary hypoplasia due to a perimandibular and perimaxillary cavernous haemangioma. **a–e** Patient's appearance and occlusion is very similar to a hemifacial microsomia anomaly. **f** The panoramic view discloses an atypical deformity of the left half of the mandible with a short ascending ramus without any gonial angle, but with a very long coronoid and a long and deformed condylar process. The latter reaches further laterally than the one on the normal side. **g** On a tracing of the panoramic radiograph (with the courtesy of H. Sailer), the difference between the left and right halves of the mandible becomes very obvious when the normal hemimandible is projected over the abnormal one

eral direction, so much so that there is a 2 cm difference between the length of the right and left mandibles, measurable on the panoramic view. That radiograph also disclosed that the articular eminence and the fossa are different to normal. The fossa seems large and flat. The eminence is very big and strong. It is 2 cm more laterally placed than on the normal right side. The maxillary sinuses do not differ very much.

The tracing of the panoramic view permits reconstruction of the abnormality of the left side to what it would be if it were normal in shape as on the other side (Fig. 20g).

The TMJ tomograms are unfortunately missing.

The mandible in *p.a.* projection (Fig. 20h), with the mouth open, shows the typical severe deviation to the hypoplastic side. The ramus seems rather thick. It also discloses that the maxilla is deformed and higher up on the same side than on the normal side. Furthermore, it shows that there is a remarkable difference in the transverse dimension between the two halves of the base of the skull, the normal right being shorter than the left. According to the panoramic view this must mainly be in

the area of the middle cranial fossa. In the angle and horizontal ramus region there is a rather large translucency, obviously after removal of the two impacted molars.

The lateral cephalogram disclosed that a large amount of the mandibular base on one side was missing while the other was of normal size and shape. The upper front teeth were severely protruded while the whole mandible was in a retromandibulism position. The chin too was retruded.

Provisional diagnosis. Hemifacial hypoplasia of the left side with severe deformity of the left mandibular and the left maxillary side, of uncertain origin.

Treatment Plan. Rotation of the lower half of the facial skeleton and advancement of the mandible and additional chin advancement. By a Le Fort I mobilization and bilateral sagittal splitting procedure of the rami, and sliding chin advancement the desired goal should be achieved. In a second stage lyophilized cartilage onlays should be placed subperiosteally in the area of the

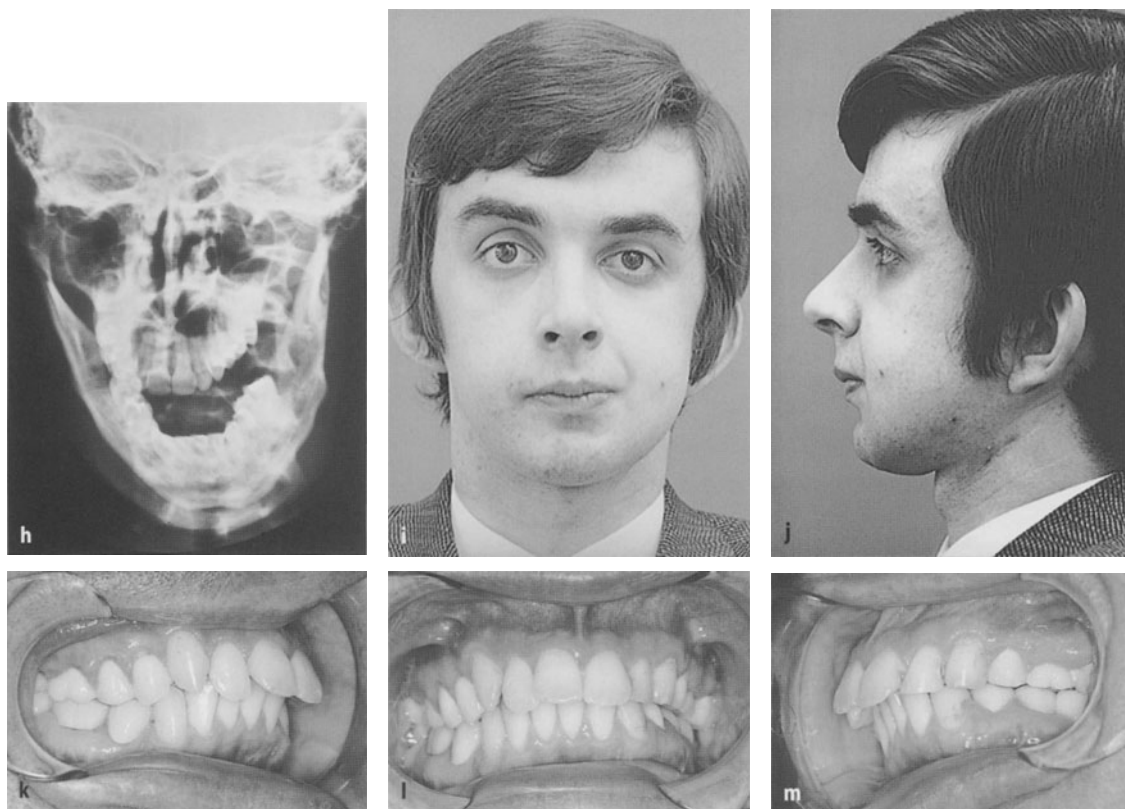


Fig. 20h-m. Hemimandibular and hemimaxillary hypoplasia due to a perimandibular and perimaxillary cavernous haemangioma. **h** The mandible *p.a.* projection at maximum opening shows a clear difference in vertical length of the two ascending rami. It also detects the severe difference of the two halves of the base of the skull in their lateral extension. **i, j** Patient's appearance 3 years after corrective surgery. **k-m** Patient's occlusion 5 years after surgical correction

facial flatness at the posterior body and in the ascending ramus and zygomatic region of the left side for additional contour correction.

Actual Treatment. The impacted teeth in the mandible were first removed without any problems. Six months later the rotation of the lower half of the facial skeleton was commenced with the sagittal splitting of the ramus on the normal right side. There were no problems. Then the left posterior body and angle area was freed from the periosteum on the lateral aspect. In the concave angle area, quite suddenly, massive venous-type bleeding occurred. There was no possibility of stopping the bleeding with artery forceps. Therefore an extraoral approach was made below the angle for better access to the bleeding. With artery forceps placed almost as high as the base of the skull it was impossible to stop the bleeding. A long strip of vaseline gauze was used to pack the large cavity behind the ascending ramus in order to stop the bleeding. The oral incision was closed and the right

split ramus was fixed in its original position with a circumferential wire and intermaxillary fixation. The strip of vaseline gauze was shortened after 2 weeks, every second day and finally removed 3 weeks postoperatively, without any further bleeding.

Fourteen weeks after that intervention, for a second time a Le Fort I mobilization and a bilateral splitting of the rami were planned. This time the abnormal left side was operated upon first and again commencing with the mandible. There was no problem in the buccal and posterior ramus area. But when the periosteum was elevated on the lingual side above the lingula, massive bleeding occurred again. Knowing that it could not be stopped by ligation or coagulation, again a strip of vaseline gauze was used to stop the bleeding by packing the area between the upper half of the mandible and the maxilla. Then the sagittal splitting of the ramus on the left side was carried out without any further problems. Next, the sagittal splitting procedure was performed on the right side and the mandible was repositioned into

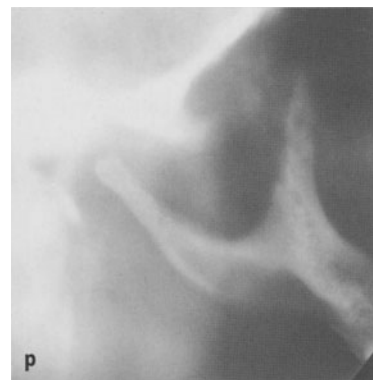


Fig. 20n–p. Hemimandibular and hemimaxillary hypoplasia due to a perimandibular and perimaxillary cavernous haemangioma. **n–p** Case of similar mandibular deformity, also due to a perimandibular haemangioma, but presenting an even more severe and hazardous problem at surgery

the desired occlusion. The fragments were secured in their new position by a circumferential wire on each side followed by intermaxillary fixation for 6 weeks. The soft tissues were closed and the end of the gauze strip was led into the mouth between the sutures. It was again shortened at intervals and finally removed after 3 weeks. No Le Fort I mobilization was performed because of the too great risk of very severe bleeding around the maxilla.

Three months later the chin asymmetry was corrected by a sliding osteotomy procedure and thin slices of lyophilized human cartilage, cut with a dermatome, were placed subperiosteally along the flatness in the ascending ramus area as well as over the horizontal part of the left mandible. The left-sided flatness still remaining was corrected. The postoperative course of both interventions was uneventful and finally a good result was achieved in the profile as well as in the occlusion (Fig. 20i-m).

Discussion. A congenital cavernous haemangioma around the ascending ramus, condyle and maxilla can obviously produce a clinical picture which resembles a hemifacial microsomia syndrome deformity. It was obvious on the panoramic radiograph that the condyle on the affected side was absolutely abnormal. It was only like a fingertip, not really even as one would expect after rheumatoid arthritis. This destruction of the condyle obviously did not permit normal growth and development of the ascending ramus.

The surgery of such a case can be quite hazardous. Before this case we had had an even more severe problem with another mandibular asymmetry case of the same origin (Fig. 20n-p). It was of the same type but much worse in its deformity. It was also planned to cor-

rect the asymmetry by a bilateral sagittal splitting procedure. A very experienced coworker of mine who was responsible for our cancer surgery, ran into great problems. With the first incision in the vestibular mucosa, massive bleeding caused great difficulties. There was no way of stopping it. He packed the oral incision and used a submandibular approach to ligate the external carotid artery and the veins. The bleeding did not stop. He extended his submandibular incision upwards preauricularly in order to ligate the bleeding vessels. No effect. He called me into the operating room as he wanted to ligate the internal carotid artery. I told him it would not help. But we ligated the internal carotid artery temporarily. We did it very carefully and the bleeding recommenced in spite of the ligation, as soon as the packing was removed. The only way to save that patient's life was to leave the packing until all the bleeding vessels were obliterated. It took 3 weeks for this to occur.

Nowadays, one should always request angiography and scintigraphy in all cases in which such a peculiar shape of hemimandibular anomaly is observed, as this type of anomaly cannot be explained by hypoactivity of the condyle alone. Even attempted aspiration with a syringe might help to avoid such an alarming intraoperative experience.

Conclusion. Perimandibular haemangiomas and in particular such pathology around the ascending ramus and condyle can prevent normal growth and produce a peculiar type of hemimandibular hypoplastic anomaly. Although the surgical procedure seems to be almost equivalent to the correction of an ascending ramus deformity in a hemifacial microsomia case, it may become very hazardous.

10.1.5

Unilateral Condylar Hypoactivity Due to Condylar Damage by Adverse Factors in Early Childhood

It is known that various adverse factors damaging the condyle in early childhood, can lead to disturbance of development of the affected hemimandible. The earlier the damage occurs, the more severely will the development be disturbed and the more hypoplastic will that side of the mandible become. However, these various

cases can teach us a lot about the development and growth of the mandible. For that reason, a few cases are demonstrated which are of special interest regarding the influence of the condyle on the development and growth of the mandible. It is a very complex subject indeed.

Unilateral Hypoplasia of the Condyle of Uncertain Aetiology¹ (Fig. 21)

Sex and Age. Female, 14 years.

Aetiology and History. Unknown; No TMJ pain in history. At the age of 8 years the girl was referred for evaluation and possible treatment because of slight asymmetry of the mandible. No relevant family history.

Clinical Findings. At the age of 8 years there is a mildly oblique lip commissure with the left corner more lateral and higher than the right (Fig. 21a). The midline of the lower teeth corresponds nearly to the uppers at that age (Fig. 21b). At age 11, the chin prominence is a bit to the left and the left side of the face seems slightly hypoplastic, noticeable particularly in the zygomatic-cheek

region. The left eye seems clearly smaller than the right and (or because of) further back (Fig. 21c). The midline of the lower incisors is shifted to the left by the width of half an incisor (Fig. 21d).

At age 13, the oblique lip commissure is now more obvious and its left corner clearly higher than the right. The prominence of the chin is now over to the left by 10 mm. The left-side facial hypoplasia is now more recognizable as well as the vertical facial shortness on the left side (Fig. 21e). There is now an easily noticeable oblique occlusal plane (Fig. 21f). At the age of 14 the asymmetry is even more apparent.



¹ I am grateful to Prof. Paul Stöckli for permitting me to use this case of his.

Fig. 21a–f

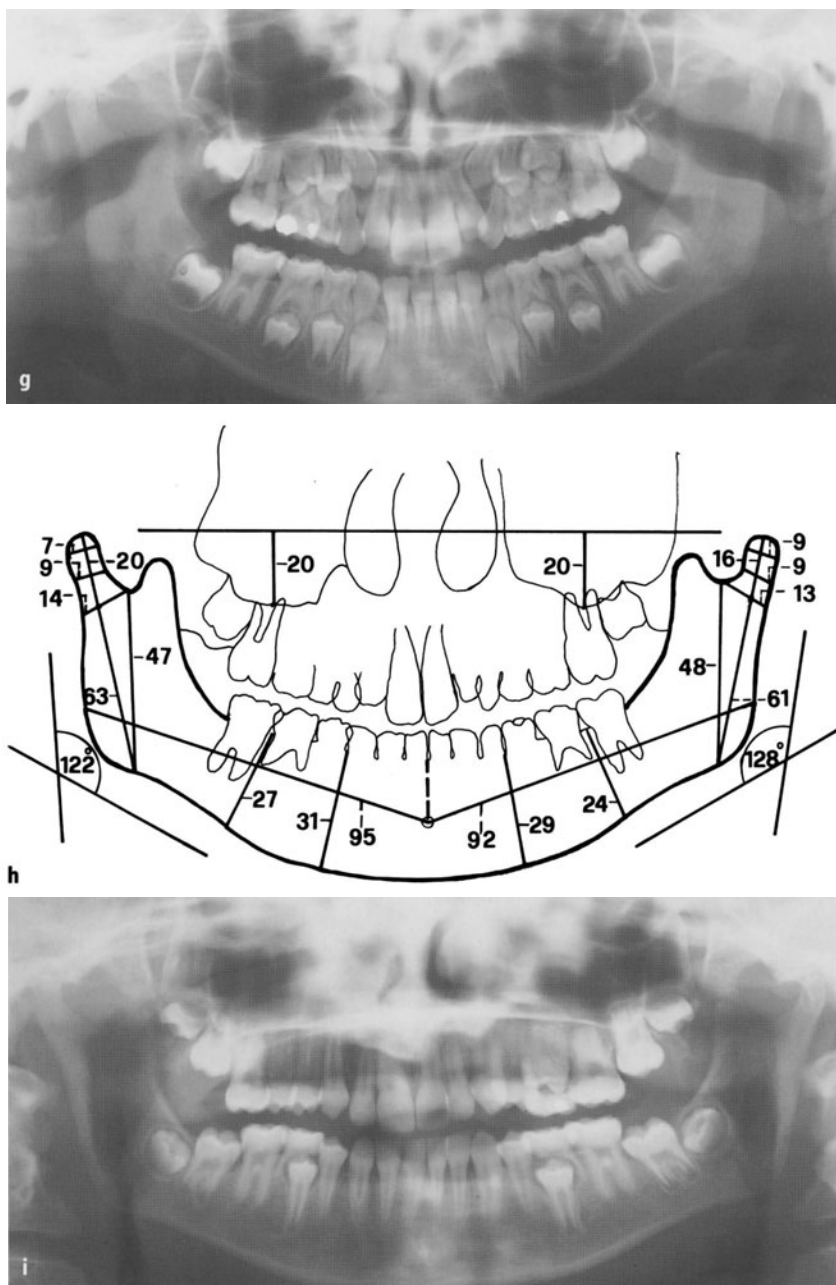


Fig. 21a–i. Unilateral hypoplasia of the condyle of uncertain aetiology. **a, b** At the age of 8 years a slight facial asymmetry is noticeable and the midline of the lower teeth is to the left by 2 mm. **c, d** At the age of 11 years the asymmetry of the face and of the occlusion is more pronounced. **e, f** At age 13 years the girl's appearance now clearly shows the consequences of hemimandibular hypoplasia, including an oblique occlusal plane and an ipsilateral hypoplasia of the zygomatic complex. **g** The panoramic view at age 8 years does not permit a clear diagnosis right away. **h** The measurements of the tracing of the panoramic radiograph, taken at age 8 years (**Fig. 21g**) detect a slight shortness of the left horizontal and ascending rami. **i** The panoramic view at age 11 already permits diagnosis of the signs of a slight mandibular hypoplasia on the left side

Radiographs. The panoramic view at the age of 8 years (Fig. 21g) discloses only a very slight difference between left and right sides. A decision could not be made as to whether or not the left ascending ramus is a bit shorter than the right and its condylar neck too short or if the right shows signs of a commencing elongation with a slightly smaller condyle and longer neck than on the left.

The measurements of the tracing made the situation clearer (Fig. 21h): the left ascending ramus is 2 mm shorter than the right, the left articular process is 4 mm shorter than the right and the left horizontal ramus is 3 mm shorter than the right.

At age 11 the situation became clearer, also in the panoramic radiograph (Fig. 21i): the left condyle and ramus are slightly hypoplastic and shorter than those on the opposite side and there is a shallow antegonial notch on the same side with the horizontal ramus a little less high in that area. At 14 the situation is clear (Fig. 21j), particularly in the panoramic view in the closed teeth position: the left side hypoplasia seems obvious, with a hypoplastic condyle. There is clearly an oblique occlusal

plane. The right condyle also seems rather small with a rather long slim condylar neck. Its angle seems within the range of normal while on the left side the antegonial notch is more pronounced.

TMJ tomograms at age 14: The left condyle is slightly hypoplastic and has a rather short neck. There is no clear sclerosis recognizable. The right condyle and neck are within the normal range. However, since no TMJ tomograms were taken during the various stages of growth, the one taken at age 14 is not of great interest regarding development of the problem. Tomograms taken at age 8 and 11 would probably have told us whether the condylar hypoplasia was then diagnosable.

Cephalometric p.a. projection (Fig. 21k, l): At age 8, the left ascending ramus seems slightly less high than the right. The left floor of the nose is slightly higher than on the right. These signs of asymmetry are even more obvious at age 13. At that age, a clear shortness in vertical height of the maxilla on the left side cannot be overlooked. This is also true for the higher level of the left floor of the nasal cavity. But in addition there is a slightly pronounced antegonial notch visible on the left side.

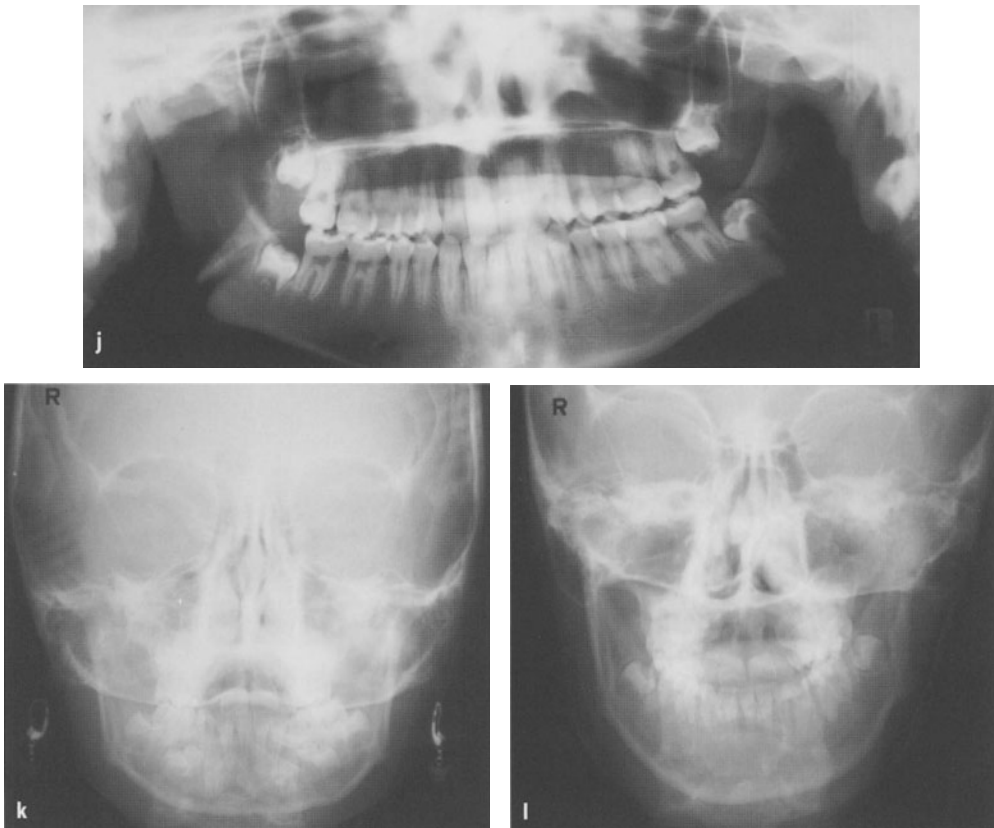


Fig. 21j–l. Unilateral hypoplasia of the condyle of uncertain aetiology. **j** The panoramic view taken at age 14 leaves no doubt. All necessary signs of a left mandibular hypoplasia are now clearly visible. **k, l** The cephalogram in p.a. projection already discloses at the age of 8 a minor difference between left and right ramus height and position of the floor of the nose. At the age of 14 the signs are clearer

The midline of the lower incisors was slightly to the left of the facial midline at age 8. At age 11 it was to the left by almost the width of an incisor and at age 13 the midline had moved slightly more to the left and the lower front teeth were tilted to the right.

Mandible p.a. at maximum mouth opening, at age 14: The left ascending ramus is extremely thin and short. The right ramus is also thin and bowed slightly outwards with elongation of the condylar neck (Fig. 21m). The difference between left and right sides is more recognizable in this projection than in all the others. That might also have been the case at age 8 years.

Hand radiographs: At the age of 14, the epiphyseal plates of the radius and the ulna bones were almost closed and there was complete closure of the finger epiphyses.

Scintigraphy. Scintigraphy with Technetium-99-DPD, performed by the Department of Medical Radiology, Clinic and Polyclinic for Nuclear Medicine (Head: Prof. G. von Schulthess) shows a clear difference in activity between right and left sides (Fig. 21n): There is still some, but very little, or almost no, signs of activity in the left condyle but clear activity in the right condyle. Diagnosis: Pronounced growth activity in the right mandibular condyle. Compared with the left condyle it showed 41.5% more activity.

Provisional Diagnosis. Either left sided condylar hypoplasia of unknown origin or H.E. of the right side or both.

Treatment Plan. Discuss the situation with the patient and her parents. Either suggest waiting until the patient herself requests improvement, or recommend performing a vertical rotation of the lower half of the facial skeleton plus chin transposition after growth has ceased, in order to achieve facial symmetry.

Treatment Performed. At present, none.

Discussion. This was not an easy case to diagnose. The facts were, that already at the age of 8 there were possible signs of facial asymmetry due to vertical shortness on the left side. At that age there were no skeletal signs of a right sided H.E. or H.H. The asymmetry became more obvious until 14 years of age, when general growth of this patient had already finished. Scintigraphic investigation at that age revealed that there was clearly almost no growth activity in the left condyle but there was 41.5% more on the right side. That might suggest that the right condyle suffered from growth hyperactivity. If there is nothing on one side then some activity on the opposite side represents a high percentage increase (Fig. 21n). According to the shape of the right hemi-



Fig. 21m, n. Unilateral hypoplasia of the condyle of uncertain aetiology. **m** In the mandible p.a. projection at maximum mouth opening at age 14, the hypoplasia of the left mandible cannot be missed. **n** Scintigraphy at age 14 discloses clear hypoactivity on the left side compared with the right side

mandible there was definitely no developing H.E. The elongation of the right condylar neck might have suggested a developing H.E. However, there was no clear stretching of the angle and the lower teeth midline had not moved further to the left. So, the existing condylar growth activity on the right side at age of 14 may still have been part of normal mandibular growth while the left sided inactivity may be due to condylar hypoactivity with the condylar hypoplasia syndrome as a consequence.

If it were a H.E. of the right side only then there could not be a lack of downward growth of the maxilla on the left side. That fact is definitely only due to the lack of length of the left ascending ramus. If it were an additional H.E. on the right side, the chin and the midline of the lower teeth should have shifted much more over to the left side.

For the differentiation as to whether that condylar hypoactivity was of congenital or very clearly postnatal origin we have to find a sign which is only seen in one of the two. The early lack of vertical downward growth of the maxilla as well as the midline deviation of the lower teeth to the affected side and the antegonial notch, is found in both aetiologies of condylar hypoactivity. But the fact that at the age of 8 the left condyle was almost equal in size to that of the right and only the left ascending ramus was slightly shorter, suggests that this condylar hypoplasia is not of congenital origin. This diagnosis is also supported by the fact that 6 years later the hypoplasia of the condyle was very obvious. A congenitally hypoplastic condyle would already have appeared as such before the age of 8 years.

If the p.a. projection of the mandible in the maximum opening position had been requested at age 8 years, the diagnosis would probably have been clear at that age already, as that projection taken at 14 years showed the asymmetry better than did any other radiograph.

Conclusion. The history of this 14-year-old girl with clinical and radiological findings from 8 to 14 years of age showed clearly the hypoplasia of the left condyle and consequently of the left mandible and the left maxilla. The fact that the right and left condyles were rather similar in shape and size at the age of 8, and that the left condyle became rather hypoplastic by 6 years later, indicates it was more probably of postnatal origin than intrauterine developmental.

The cellular activity in the right condyle at the age 14, found on scintigraphy, did not prove the existence of a H.E., although general skeletal growth had already practically ceased according to the already existing epiphyseal fusion in the fingers. Mandibular growth can still proceed slightly beyond the general cessation of skeletal growth. The lack of cellular activity in the hypoplastic condyle was no surprise in view of its hypoactivity. On the other hand, H.E. would have shifted the mandible markedly to the contralateral side. This was not the case. For all these reasons, this case had to be diagnosed as a left sided hemimandibular hypoplasia due to a postnatally acquired condylar hypoactivity of unknown origin.

The radiographs of this case demonstrated clearly that several standard projections should routinely be requested. The p.a. projection of the mandible in the maximum open position is a very important one for mandibular asymmetries, but probably rarely requested by orthodontists.

Treatment had to be surgical correction of the asymmetry by rotation of the masticatory skeletal complex. It might have been wise to have performed a high condylectomy very early, thus eliminating the negative growth influence of the unilateral condylar hypoactivity, thereby permitting the functional matrix to allow the affected mandible to develop normally. Then the maxilla could also have grown downwards as on the normal side. This would also have been the case if a costo-chondral graft had been inserted as early as possible.

Unilateral Hemimandibular Hypoplasia After Complete Unilateral Posttraumatic Ankylosis in Early Childhood (Fig. 22)

Sex and Age. Male, 15 years of age.

Aetiology and History. At the age of 1 year the boy suffered burns to his neck and 6 months later he fell out of bed and sustained a fracture of the neck of his left femur and a fracture of his mandible. He developed ankylosis of the left TMJ for which he was operated upon at the age of 2, but 1 year later there was complete ankylosis again. He had to undergo several free skin grafts for the correction of the burn scars of his neck. At the age of fifteen he was referred for the release of the TMJ ankylosis and correction of the resulting deformity.

Clinical Findings. Typical external appearance of an asymmetrical birdface and many scars on his neck following skin grafting (Fig. 22a–c). The right mandibular area is flat as is the chin region of that side. The prominence of the chin is far to the left and retruded. Motor activity and sensation in the facial soft tissues are normal. There is complete lack of mouth opening (Fig. 22d). The teeth in the maxilla on the left side are crowded. The lower anterior teeth are protruding (Fig. 22e–g).

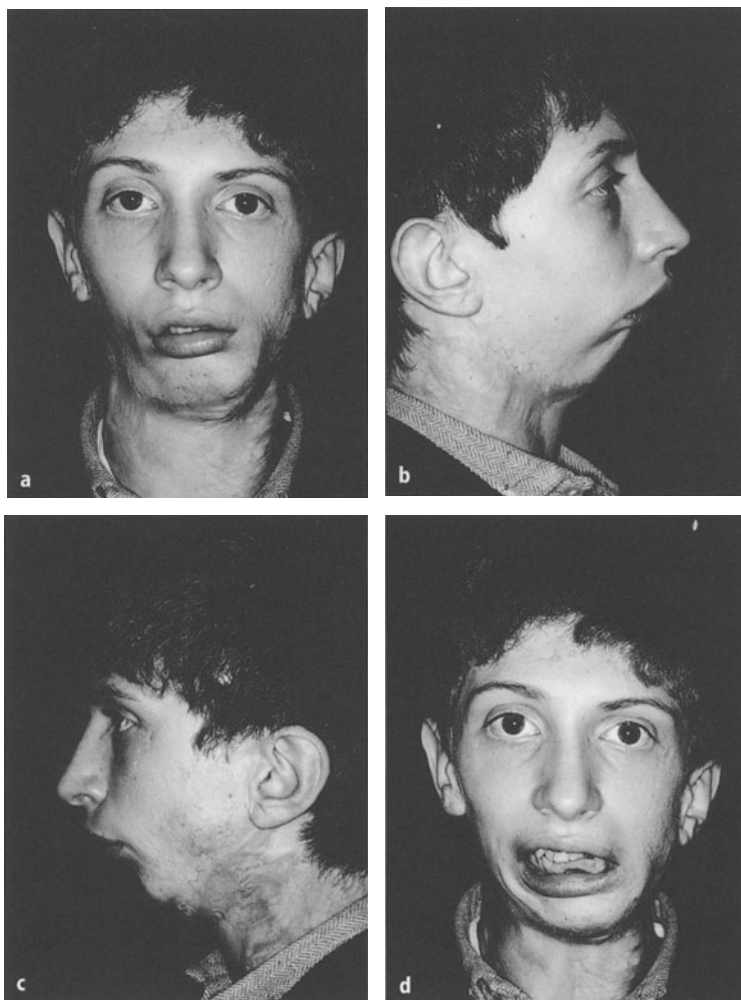


Fig. 22a–d. Case of unilateral hemimandibular hypoplasia after unilateral posttraumatic complete ankylosis in early childhood. **a–d** Patient's appearance at age 15 years and 5 months in the resting position and with no mouth opening at all, and both profile views

Radiographs. The panoramic view (Fig. 22h) shows the left-sided bony ankylosis with a bulky bony mass in the left TMJ region. There is the typical associated deformity of the ascending ramus, the angle and the mandibular body with shortness of the ascending ramus and body on the same side, a well-defined antegonial notch, an almost 90° angle of the jaw and a finger-like coronoid

process. The right side of the mandible seems normal in shape, length and height. The symphysis area resembles a notch. The teeth are crowded, some are impacted or not yet erupted. Tooth 46 (according to the international system for identification of teeth) has apical granulomas. The base of the maxilla is oblique with vertical shortness on the affected side.

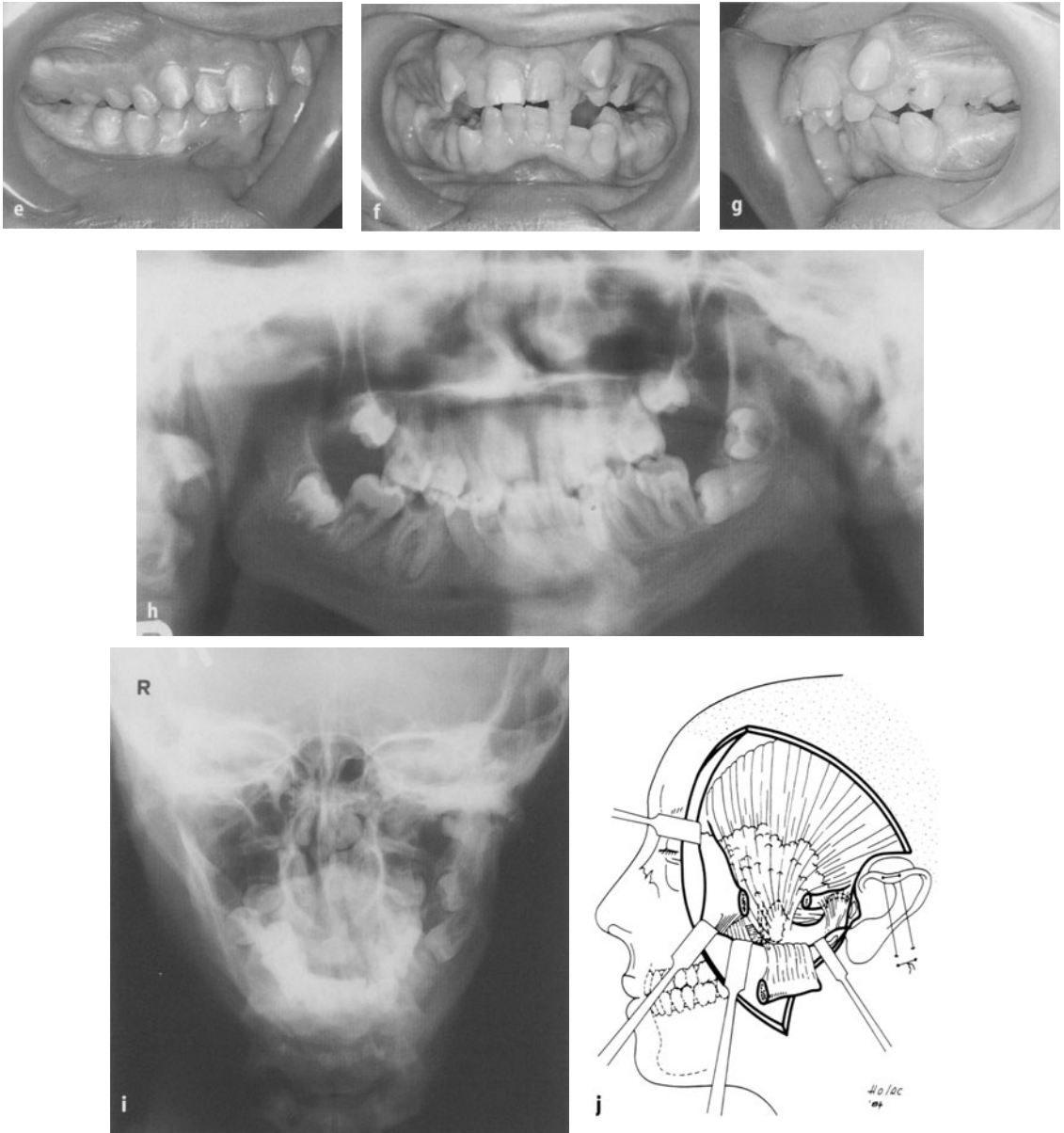


Fig. 22e–j. Case of unilateral hemimandibular hypoplasia after unilateral posttraumatic complete ankylosis in early childhood. **e–g** Patient's dental and occlusal situation before surgery. **h, i** The panoramic and the mandible p.a. views disclose the complete bony ankylosis on the left side with severe deformity of that hemimandible with crowding and impaction of several teeth, but an absolutely normally developed right half of the mandible. The posterior rim of the affected ramus is directed towards the glenoid fossa. **j** Diagrammatic illustration of retrotragal-temporal approach to the TMJ region in cases of massive posttraumatic ankylosis

P.a. mandible in the closed position (Fig. 22i): On the right side the ramus seems normal, but the body seems tilted dorsally; trabecular structure is normal. The left ramus and body seems short and wide with some impacted molars. Near the cranial end of its ascending ramus an abnormal bony mass projects into it and overlaps its medial surface. Its lower border is tilted outwards producing a step with the right mandibular body in the chin region.

TMJ tomograms: The right TMJ–ramus tomograms showed a normal configuration and position of the condyle, ramus and angle area. However, the ramus seemed shorter than normal. The left side showed the picture of posttraumatic ankylosis with a long finger-like coronoid process, shortness of the ascending ramus, an almost rectangular gonial angle and a very pronounced antegonial notch.

Lateral cephalometric view (Fig. 22u): Typical severe birdface appearance with bimaxillary alveolar and den-

tal protrusion, very extreme in the mandible. In the TMJ area only a shapeless bony mass can be recognized.

Provisional Diagnosis. As follows:

1. Left sided post-traumatic TMJ ankylosis, from the age of 1½ years, in a 15-year-old boy.
2. Asymmetric birdface.
3. Large scars on the neck after burns and multiple subsequent skin grafts.

Treatment Plan. As follows:

1. Release of the ankylosis in order to achieve normal mouth opening (Fig. 22j) followed by opening exercises for one year.
2. Presurgical orthodontic treatment.
3. Removal of the impacted teeth.
4. Correction of asymmetric birdface appearance half a year later (Fig. 22k,l).
5. Postsurgical orthodontic treatment.

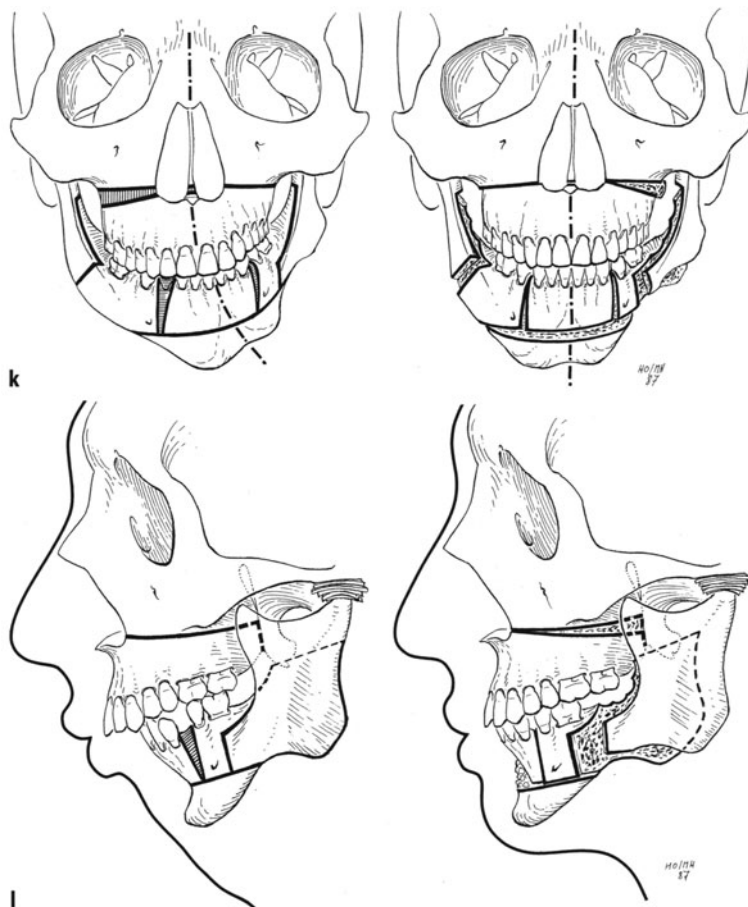


Fig. 22k, l. Case of unilateral hemimandibular hypoplasia after complete unilateral posttraumatic ankylosis in early childhood. **k, l** Diagrammatic illustration of the planned surgical correction, in front and in side view

6. Approximately 1 year after the main correction, removal of the wires in the chin region and, in addition, a double-step chin advancement and contour correction if necessary.

Actual Treatment. In this case the treatment was as follows:

1. At the age of 15½ years, the left-sided ankylosis was released and the elongated coronoid process cut off. Our standard procedure was used (M. Farmand and H. Obwegeser 1981): Via a retrotragal-temporal approach, the whole bony mass in the TMJ area and on

the lingual side of the ascending ramus was excised and the coronoid process was cut at its base. The musculature was gently stretched with a mouth opening instrument to produce normal mouth opening during surgery (40 mm interincisal distance). Next, a package of slices of lyophilized cartilage was placed into the gap in the TMJ area.

Postoperatively the patient had to exercise hard with wooden spatulae to achieve a mouth opening of 40 mm interincisal distance, as his chewing musculature tended to shorten again as it had not grown longer during the normal growth period. In addition,

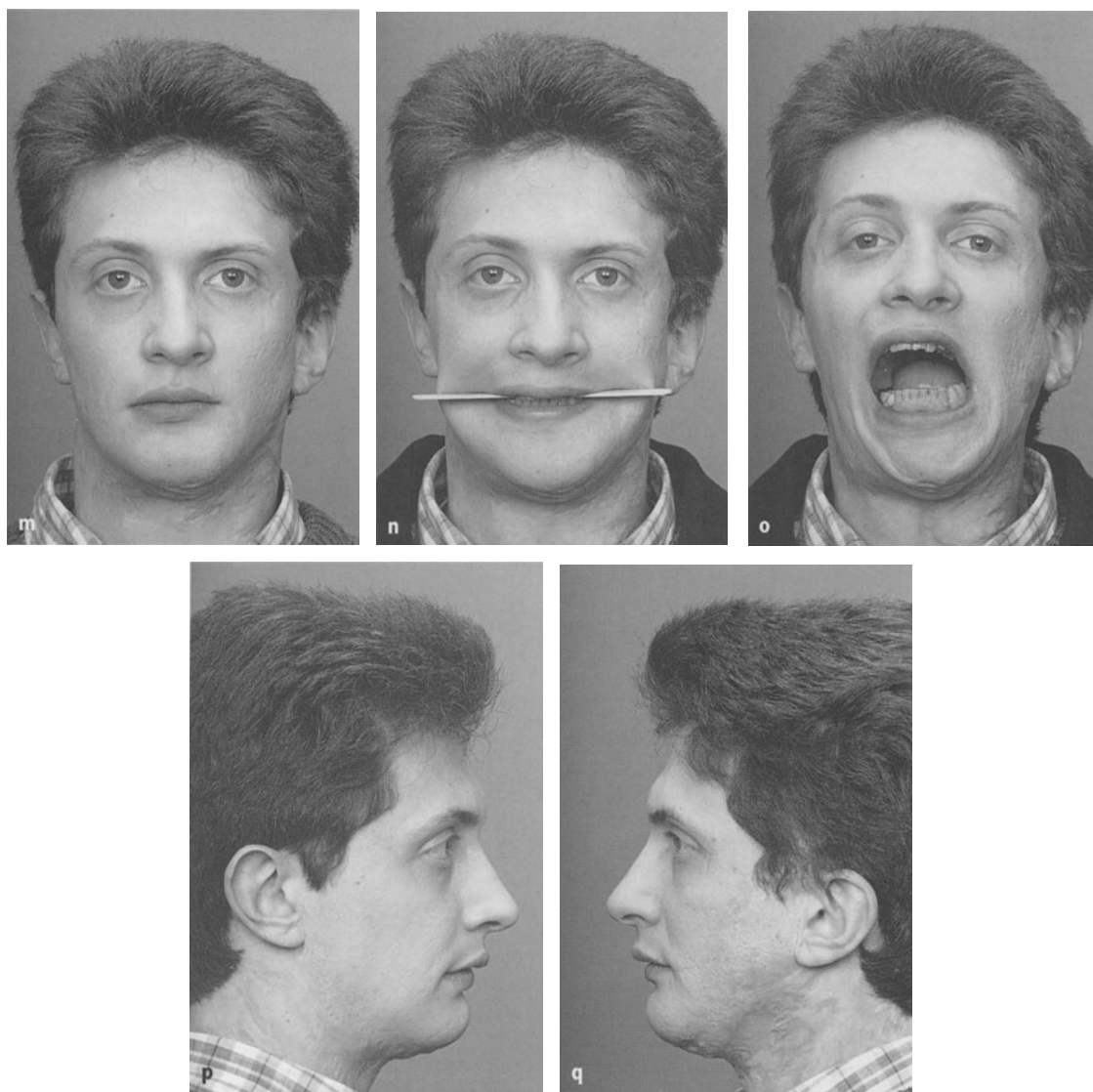


Fig. 22m-q. Case of unilateral hemimandibular hypoplasia after complete unilateral posttraumatic ankylosis in early childhood. **m-q** Patient's appearance 1 year after final treatment in front and profile views and at maximum mouth opening and horizontal occlusal plane

a type of monobloc appliance maintained the opening during the night. After one year of exercising there was no further tendency for a reduction in the gained 40 mm mouth opening. The presurgical orthodontics could now begin.

2. Presurgical orthodontics for about 3 years.
3. Four and a half years after the release of the ankylosis the impacted teeth 18, 28, 37, 38 were removed.
4. At the age of 20, 5 years after the release of the ankylosis and 5 months after removal of the impacted teeth, the skeletal deformity was corrected, again following our principle: *"Make an asymmetrical facial skeletal deformity first into a symmetrical one. Then it is either already corrected or it becomes easier to correct"* (Principle Nr. 41). The following standard procedure was performed: Le Fort I osteotomy mobilization for cranial repositioning of the maxilla on the normal right side by 6 mm and lowering the left side also by 6 mm for correction of the vertical discrepancy; extraction of the first premolar on the normal side of the mandible including a vertical osteotomy between the 45 and 43. Then mobilization of the lower anterior alveolar segment with the four incisors and canines, bilateral sagittal splitting procedure

of the rami, on the short side using a slightly elongated split with the vertical lateral cortical cut at the first molar level, vertically down to the inferior border. This was followed by detachment of the lower border of the chin with a muscle pedicle. Since the normal side was now shortened by the width of the extracted premolar, both sides could be shifted forward making two mandibular bodies of equal length. The detached lower border of the chin was then used to bridge the vertical osteotomy gaps produced by the mobilization of the anterior alveolar segment. Plates and wires were used to stabilize the six sections of the mandible in the preoperatively planned occlusion. Eight weeks postoperatively the patient was referred once more to his orthodontist.

5. One year later, the osteosynthesis materials were removed and a double step chin advancement, pedicled on the musculature was performed to increase the chin prominence, and slices of lyophilized cartilage were onlaid subperiostally on both sides of the mandible to improve the contour. Deep frozen cancellous bank bone was used to fill the large step between the two-step chin advancement and the anterior surface of the lower alveolus.

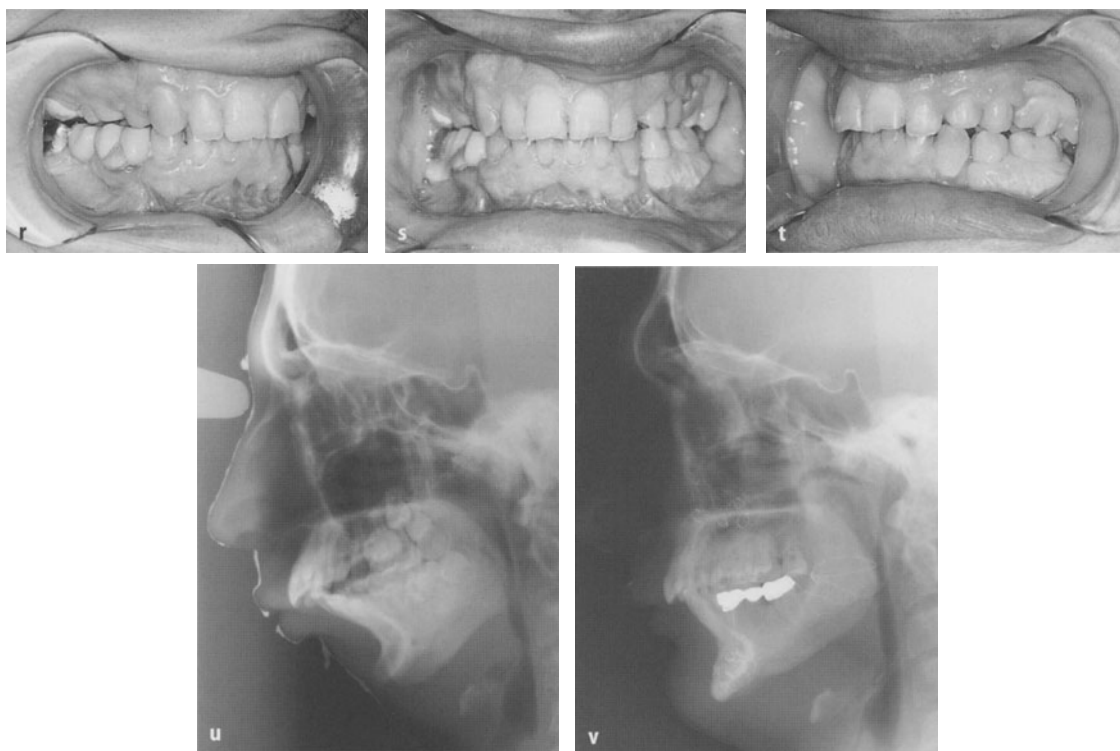


Fig. 22r–v. Case of unilateral hemimandibular hypoplasia after unilateral posttraumatic complete ankylosis in early childhood. **r–t** Patient's occlusal situation 1 year and 4 months after corrective surgery. **u, v** Lateral cephalograms before surgery and 1 year and 1 month after double-step chin advancement, in addition to the previous correction of the main skeletal deformity

Result. I last saw the patient 2 years after the correction of his facial skeletal deformity, which was 7 years after the release of the ankylosis. The spontaneous mouth opening was 33 mm with some deviation to the formerly ankylosed left side. The facial contour has remained very gratifying with no late postoperative change at all (Fig. 22m–v).

Discussion. In a case with unilateral posttraumatic ankylosis due to trauma at the age of 1½ years, with no mouth opening at all, a very pronounced unilateral hypoplasia of the mandible resulted. The height of the ascending ramus was not much less than on the normal side except for the loss of the condyle and its neck. Therefore, the discrepancy in vertical height was not very striking. The unaffected half of the mandible developed and grew absolutely normally, in spite of lack of any functional movement. It is striking that the traumatic destruction of the condyle in very early childhood, followed by ankylosis, had not only produced severe mandibular asymmetry but much less vertical height discrepancy than one would have expected when compared with cases of congenital aplasia or an in infancy-acquired hypoplasia of the condyle or even more after osteomyelitic destruction of the condyle and as-

ending ramus or in cases with hemifacial microsomia. The unaffected side obviously did not need any masticatory function in order to develop normally. Whether this was due to the fact that the condyle was fully intact or due to the “functional matrix”, we do not know. Since there was no movement of the mandible, the affected side must have had the same amount of muscle stimulation as the unaffected side, since on both sides the musculature was fully intact. This would lead to the conclusion that the intact musculature alone, without any physiological function, does not act as a “functional matrix”. So it would have to be the intact condyle only which guaranteed normal growth of the unaffected side.

Conclusion. In cases with unilateral posttraumatic ankylosis, the destroyed condyle leads to severe lack of growth on the affected side of the mandible as far as the symphysis. The lack of growth affects the body of the mandible more than the ascending ramus. The undamaged side grows absolutely normally in spite of lack of function. As the musculature of the affected side produced the same functional matrix as that on the unaffected side, this fact leads to the conclusion that the intact condyle alone can guarantee normal growth of its hemimandible without any influence of function.

Unilateral Hemimandibular Hypoplasia After Unilateral Ankylosis Due to Purulent Otitis Media in Infancy and Release of the Ankylosis at the Age of 11 (Fig. 23)

Sex and Age. Male, 24 years of age.

Aetiology and History. Purulent otitis media on the right side at the age of 1½ years resulting in complete ankylosis of the right TMJ. Not only could the patient not open his mouth, but also his mandible became asymmetrical. At the age of 11 years the boy underwent an operation for the release of the ankylosis. “Good” mouth opening was achieved according to his opinion. The asymmetry of his chin grew worse. He presented for correction at the age of 24.

Clinical Findings. The patient is in good general health. His face is asymmetrical, the left side narrower and shorter than the normal right side. His chin is severely deviated to the right and retruded (Fig. 23a). He has reasonable mouth opening (25 mm interincisal distance) but with severe deviation of the mandible to the right (Fig. 23b). He says that he can eat everything. For that reason he refused to have an additional operation to improve mouth opening.

There is a severely oblique occlusal plane (Fig. 23c). His chin is almost touching his neck and deviates to the

right, when looking at his face from below (Fig. 23d). Both his profile views show the severely retruded chin and lack of vertical height as the result of his mandibular-related short face (Fig. 23e, f). The lower tooth midline is shifted to the short side by almost the width of a lower incisor (Fig. 23g) and the occlusal plane is ascending towards the right side. All teeth are present and in rather good condition.

Radiographs. The panoramic view shows a severely deformed asymmetrical mandible and an oblique occlusal plane (Fig. 23h). The left mandible seems normal in shape and length in all regions, except that there is reduced height of the body in the preangle region, producing a shallow antegonial notch and the breadth of the ascending ramus is less than normal. The shape and size of the left condyle and its neck seems to be within normal limits. The right mandible is much too short, in the body as well as in the ascending ramus. The angle is very pronounced with a deep antegonial notch. The region of the second molar looks as if that tooth had been

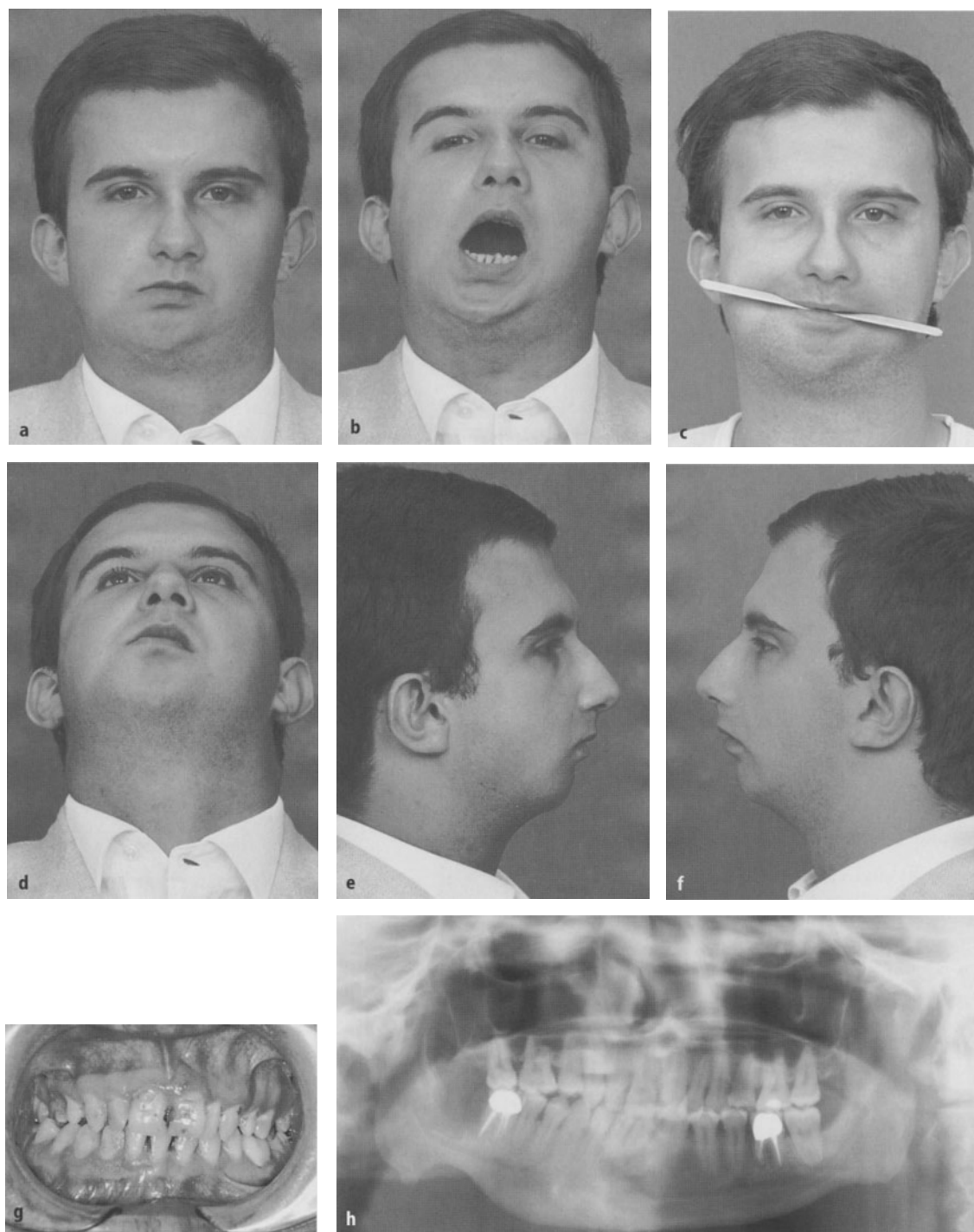


Fig. 23a–h. Unilateral hemimandibular hypoplasia after unilateral ankylosis due to purulent otitis media and release of the ankylosis at the age of 11 years. **a–g** Patient's appearance, his maximum mouth opening and severely oblique occlusal plane and his occlusal situation before corrective surgery. **h** Panoramic view demonstrating the very abnormal shape of a rather small mandible

removed surgically not too long ago. There is a very slim, finger-like coronoid process and a mass of bone in the TMJ region. Between it and the very short ascending ramus there is a strip-like defect. The latter can be clearly seen in the *TMJ-ramus tomograms* (Fig. 23i,j). The border of the defect is covered with cortical bone and it is presumed that the bony mass in the TMJ area had developed from the destroyed condyle. The condyle-neck region on the left side is normal in shape, well covered by cortical bone but rather small and thin. Because of its structural appearance it cannot be called hypoplastic. The *p.a. projection of the mandible* in the open mouth position (Fig. 23s) shows the enormous difference in vertical height between normal left and deformed right side of the mandible, as well as the maxilla. *Lateral cephalogram*: It did not show the very severe deformity of the mandible and maxilla as seen in the panoramic view and in the *p.a. projection of the mandible* in the open mouth position. There was an obvious shortness of the mandible, a small chin prominence and an almost vertical position of the upper front teeth.

Provisional Diagnosis. Severe asymmetry of the mandible due to shortness of the right body and ramus and lack of vertical height of the maxilla after an infection of

the TMJ in infancy, followed by ankylosis, which was released at the age of 11. In addition there was a very pronounced oblique occlusal plane due to shortness of the ascending ramus and maxilla on the affected side.

Planning of Corrective Surgery. As always, the occlusal situation as well as the osteotomy sites and the necessary amount of movement of the maxilla and the various segments of the mandible were planned on operating models of plaster of Paris by transferring the measurements found in the graphic planning to the model operation.

Treatment Plan. 1. Presurgical orthodontics was suggested but rejected by the patient.

2. For correction of the skeletal deformity the following was planned (Fig. 23k): rotation of the lower half of the facial skeleton according to the procedure advocated for the correction of hemifacial microsomia (H. Obwegeser 1970): Le Fort I-type osteotomy followed by repositioning of the maxilla, higher on the left side and downwards on the right side with an iliac crest bone graft interpositioned to maintain the levelling of the vertical height of the maxilla. In addition, the very short right ascending ramus must be lengthened according to the inferior positioning of the maxilla on that side and

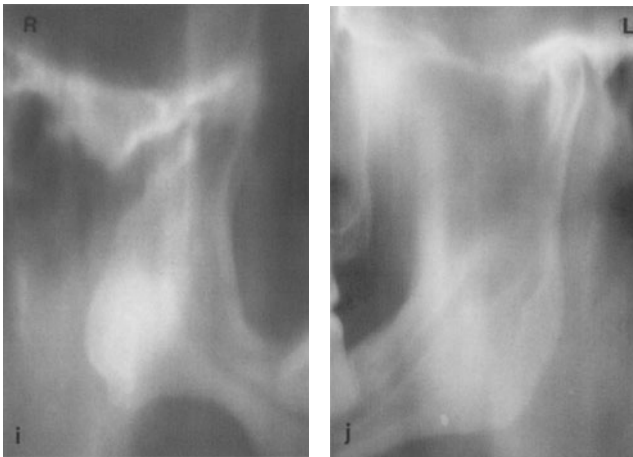
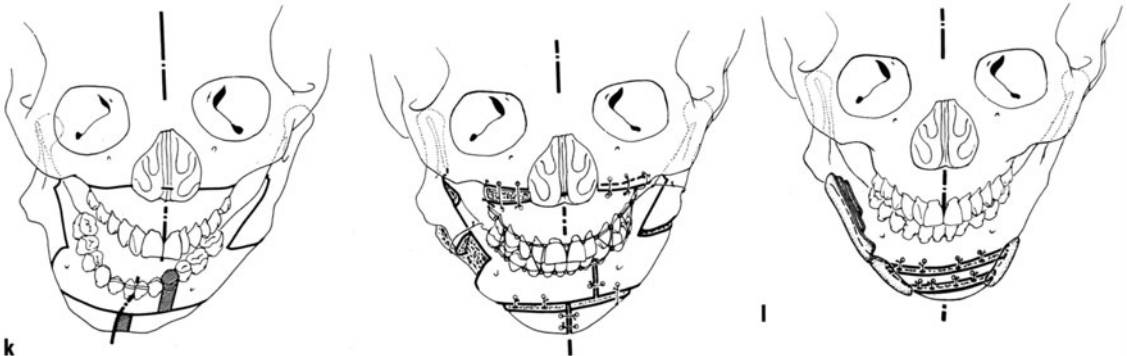


Fig. 23i-l. Unilateral hemimandibular hypoplasia after unilateral ankylosis due to purulent otitis media and release of the ankylosis at the age of 11 years. **i,j** The TMJ tomograms disclose why a certain amount of mouth opening was possible and also that in this case the length of the ramus trunk on the affected side is much less than that on the other side. **k** Diagrammatic illustration of surgical plan to produce a symmetrical facial skeleton. **l** Diagrammatic illustration of the second surgical intervention for additional increase in chin prominence and correction of the narrow mandibular contour by onlaying of lyophilized cartilage slices



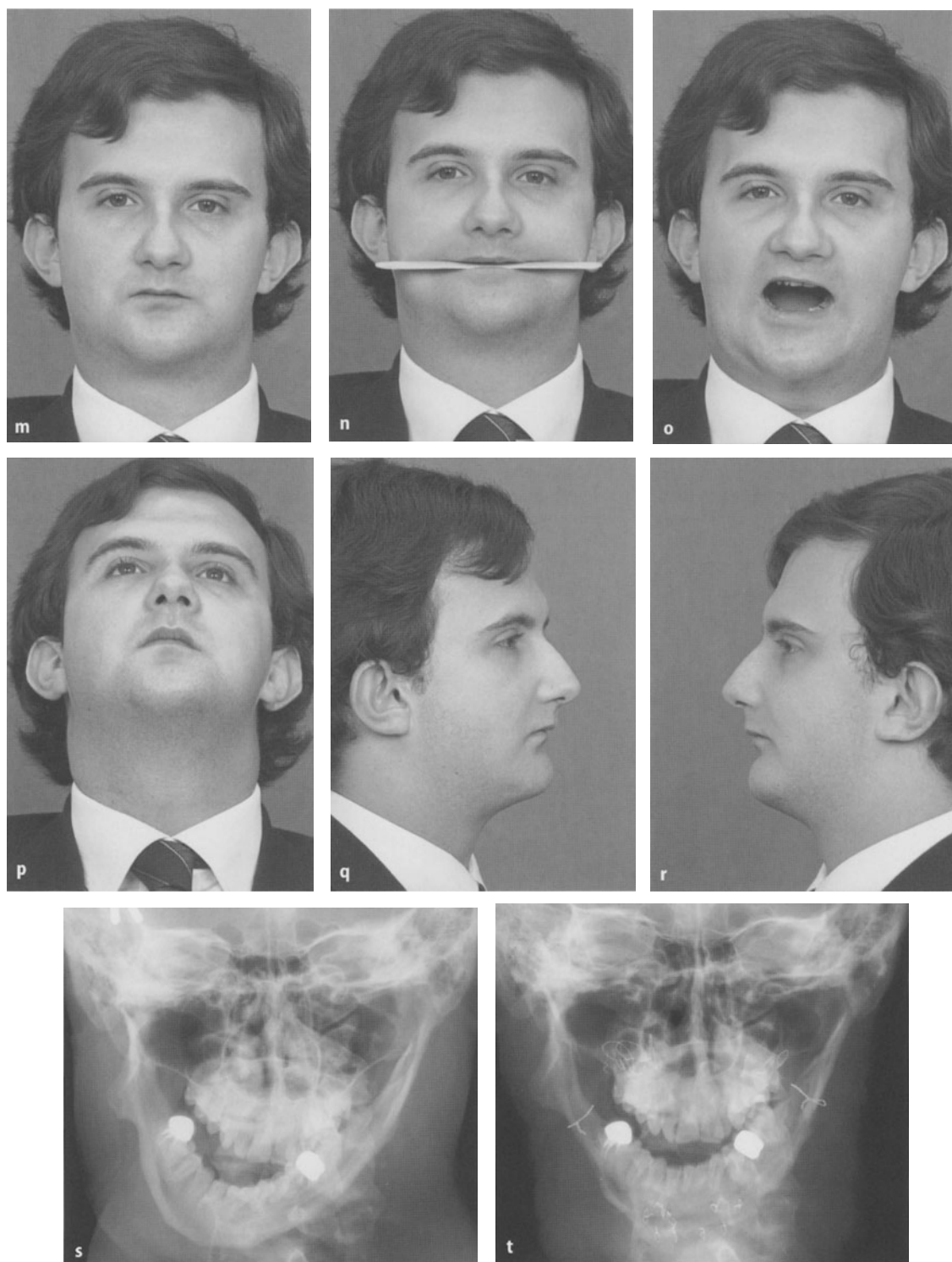


Fig. 23m-t. Unilateral hemimandibular hypoplasia after unilateral ankylosis due to purulent otitis media and release of the ankylosis at the age of 11 years. **m-r** The final result of the two-stage corrective surgery with an absolutely symmetrical facial appearance. **s,t** The two p.a. radiographs of the mandible disclose the pre- and postoperative shapes of the mandible

the whole mandible had to be moved forwards using a bilateral sagittal splitting procedure on the rami. An extraction and vertical osteotomy in the left first premolar region is necessary to increase the possibility of a larger mandibular advancement. Furthermore, a muscle-pedicle chin advancement would help to some extent to correct the retruded chin.

3. In a second operation an additional double step chin advancement and onlays of slices of lyophilized cartilage may be necessary to obtain enough chin prominence and also to correct the lateral hypoplastic areas of the mandible (Fig. 23l).

Actual Treatment. The correction was executed as planned, in two operations.

First a graft of mainly cancellous bone, measuring 6×3 cm, was taken from the right iliac crest. Next the maxilla was mobilized in the usual manner. It was repositioned 3 mm higher on the normal left side and 6 mm inferiorly on the abnormal right side. A piece of cancellous bone was wedged in the gap in the antral wall on the right side. Then the maxilla was fixed in the new horizontal position with the previously placed wires. The vestibular incision in the upper jaw was then closed.

Next the chin area was partially exposed and its lower border was cut off as far as the mental foramina, leaving the lingual musculature attached.

Then the mandible was mobilized using a long-surface ramus-body splitting procedure on the short right side and our routine sagittal splitting of the ramus on the left. After splitting the mandible on both sides the mandibular segments were properly mobilized. For that mobilization on the short right side, and in order to be able to elongate the ramus vertically, it was necessary to free the lingual surface of the ascending ramus from all surrounding scar tissues as well as the muscle sling from the lower border. Then the first bicuspid on the normal left side was extracted. A vertical osteotomy in that area permitted the right main fragment including the teeth to be shifted to the left side by the width of a bicuspid. Thereby out of an asymmetric short mandible a symmetrical one was created. Now the mandibular segments were fixed in occlusion with the upper teeth. Next, all parts of the mandible were fixed together in the new position by wire osteosynthesis after the detached asymmetric chin prominence had been cut in the symphysis region in order to make the prominence symmetrical. The mandible came forward 15 mm on each side. The surgery was followed by intermaxillary fixation for 6 weeks. One year later, a double-step chin advancement was performed and lyocartilagene homografts were added subperiostially to the still narrow lateral areas. The final result was an absolutely symmetrical face with a good chin prominence and a horizontal occlusal plane (Fig. 23m-t).

Discussion. It was assumed that the purulent ear infection in infancy had severely affected the right condyle with its neck. Ankylosis resulted. As visible on the panoramic radiograph and on the TMJ tomogram, the surgeon who did the operation to release the ankylosis did not remove the ankylosed bony mass which included the former condyle. A fairly good mouth opening was achieved. But the affected hemimandible did not grow. We knew by experience that after release of ankylosis the affected side of the mandible will start to grow again when the ankylosis is released before general growth has ceased. The younger the patient when the ankylosis release is done, the more normal will growth be found. This has also been reported in the literature (O. Kosuke et al. 1981).

We always try to remove the damaged condyle whether infection or a trauma is the cause of the ankylosis.

In this case we were unable to say that the failure of additional growth after the release of the ankylosis was due to the fact that the severely damaged condyle had not been removed or whether that side of the mandible had just failed to react, with growth, to the functional matrix, in spite of the fair amount of mouth opening gained. I believe in the necessity for complete removal of the severely damaged condyle in young patients with ankylosis. If the whole condyle-neck and upper ramus region were sclerosed because of the previous infection, then it might have been wise to have replaced it with a costo-chondral graft.

Conclusion. Purulent destruction of the condyle and neck in infancy leaves no chance for normal growth of the affected hemimandible to occur. The destroyed condyle and neck should be removed as early as possible. Normal function will then also permit further growth of the affected side. Even without a costo-chondral graft we have often experienced this. An already existing lack of growth will not be corrected with this. For that, an additional insertion of a costo-chondral graft may be necessary when the patient is still in the growth period.

In fully-grown patients, additional downwards and forwards growth of the affected side cannot be expected just by releasing the ankylosis. In these cases, nowadays, the so-called distraction osteogenesis is in favour. I have often achieved the desired vertical elongation of the short ascending ramus and the additional horizontal elongation of the body just by the use of the sagittal splitting procedure, as described in the operation report of this case.

Unilateral Hemimandibular Hypoplasia After Destruction of the Condyle and Ramus Due to Osteomyelitis in Infancy, but with Unhindered Mouth Opening (Fig. 24)

Sex and Age. Female, 19 years of age.

Aetiology and History. Normal delivery. At the age of 6 weeks she had a high fever and a purulent discharge from the left ear and TMJ region. Later, the usual childhood infectious diseases such as measles, whooping-cough, mumps. Pyelitis at age 13. Orthodontic treatment from the age of 10. Referred for surgical correction of her facial asymmetry.

Clinical Findings. Severe facial asymmetry with vertical, horizontal and sagittal shortness on the left side with all related signs such as an oblique labial commisure, flatness of the left side of the face, chin prominence shifted over to the left side, difference in vertical height of left and right sides, oblique occlusal plane, midline of lower teeth shifted over to the left, severe deviation of the mandible when opening, etc. (Fig. 24a,b). There is full opening of the mouth (42 mm intercuspal distance). There is a full upper and lower dentition except for the missing left lower second molar with a fairly good occlusion (Fig. 23c).

Radiographs. *Panoramic view* shows severe asymmetry of the mandible with a normal right side but with an extreme hypoplasia and deformity of the left ascending ramus and distal half of the right body, and a very hypoplastic condyle (Fig. 24d). The left coronoid process is finger-like and the angle area is small and severely deformed. In front of all this is a defect of the body as far forwards as the mental foramen with almost no vertical height of the left ascending ramus and posterior half of the body of the mandible. The structure shows rather dense trabeculae and no sclerosis. The mandibular canal is not visible, only the mental foramen. There is an oblique occlusal plane and base of the maxilla. The midline of the upper teeth is shifted 5 mm to the right side in relation to the nasal septum.

TMJ-ramus tomograms: Normal right side; the left side showed the same picture as the panoramic view.

P.a.mandible at maximum opening (Fig. 24e): Very severe shortness of left half of the mandible with a very

long coronoid process simulating elongation of the right ascending ramus.

Lateral cephalometric view (Fig. 24f): Very short, severely retruded mandible and chin and enormous unequal height of the two mandibular bodies. There was a 3 cm difference in the antegonial region.

Water's sinus view (Fig. 24g): Very severe vertical shortness of the left side of the maxilla with an oblique occlusal plane and reduction in size of the left maxillary sinus.

Provisional Diagnosis. Severe facial asymmetry due to extreme hemimandibular hypoplasia of the left side as the consequence of osteomyelitic destruction of the condyle, the ramus and posterior part of the mandibular body in the first few months of life.

Treatment Plan. Rotation of the lower half of the facial skeleton, reconstruction of the missing parts of the left mandible with rib grafts and reconstruction of the glenoid fossa as suggested for cases of hemifacial microsomia (H. Obwegeser 1974) (Fig. 24h). For reconstruction of the missing glenoid fossa slices of lyocartilage will be used. A long costo-chondral graft will reach from the reconstructed glenoid fossa all the way down to the chin region. It will be fixed as an onlay graft to the body and the ascending ramus for ramus reconstruction and to increase facial volume on the affected side. The chin prominence will be cut off, but left pedicled to its lingual musculature and rotated to the right side. A piece of rib will be wedged in between it and the remaining chin region for height increase on the affected side. Intermaxillary fixation and circumzygomatic-arch-suspension will be used for 6 weeks. According to the model operation the maxilla must be raised on the unaffected side by 7 mm and lowered by the same amount on the affected side with interpositioning of a bone graft. In addition the maxilla must be rotated to the right by 5 mm.

Fig. 24a–g. Unilateral hemimandibular hypoplasia after destruction of the condyle and ramus due to osteomyelitis in infancy, but with unhindered mouth opening. **a, b** Front view showing the severe facial asymmetry accompanied by a severe degree of obliquity of the occlusal plane. **c** Fairly good occlusion with deviation of the lower teeth to the hypoplastic side. **d** The presurgical panoramic view of the mandible shows the severe deformity of the left half of the mandible. **e** The preoperative p.a. projection of the mandible in the maximum opening position discloses even better the very severe deformity of the mandible caused by the shortness of the left side. **f** The preoperative lateral cephalogram discloses the large discrepancy in body height between the two sides of the mandible and the remarkable shortness of the mandible. **g** The Water's projection of the middle third of the facial skeleton discloses the amount of lack of downward growth of the maxilla



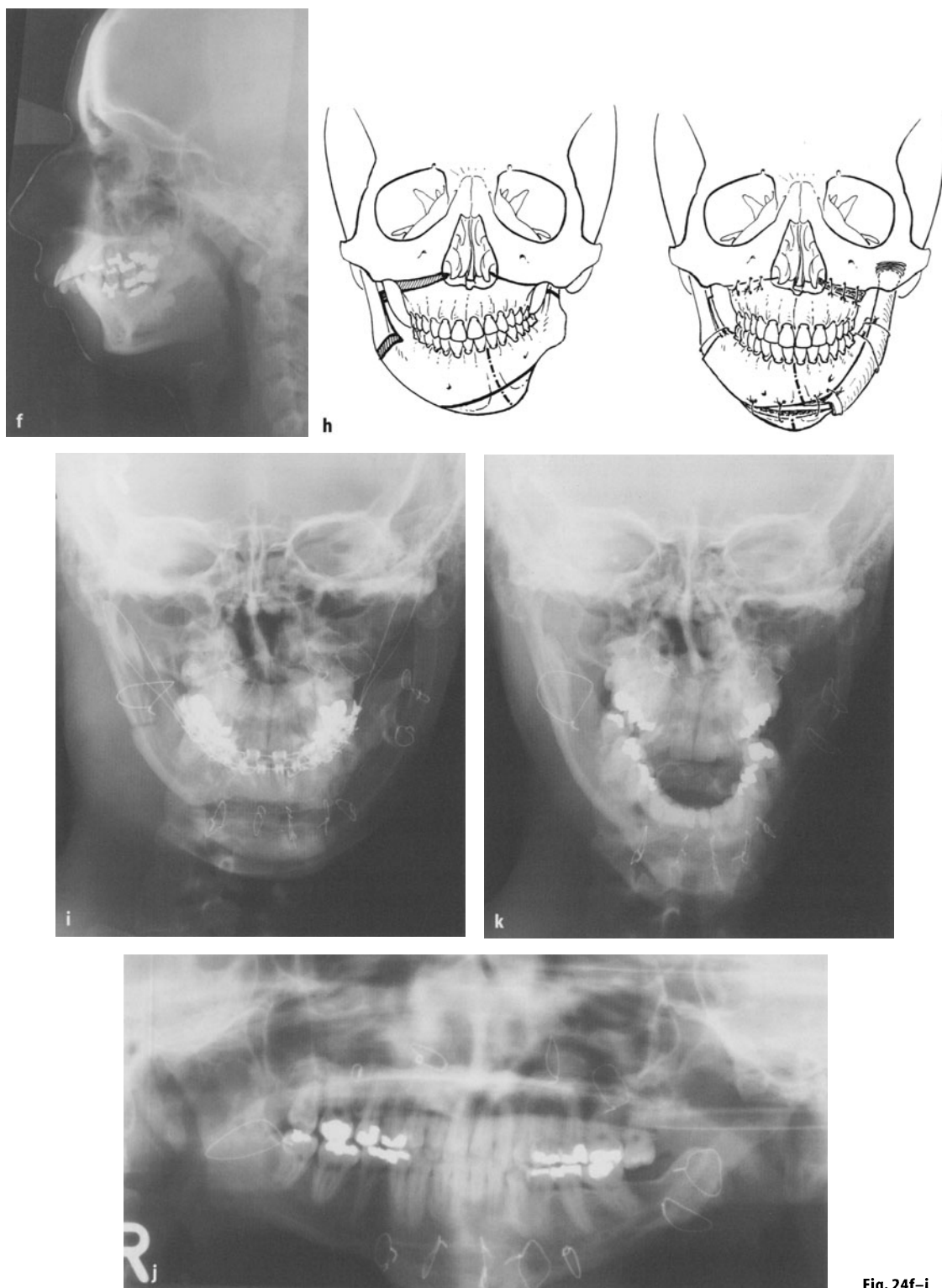


Fig. 24f-j

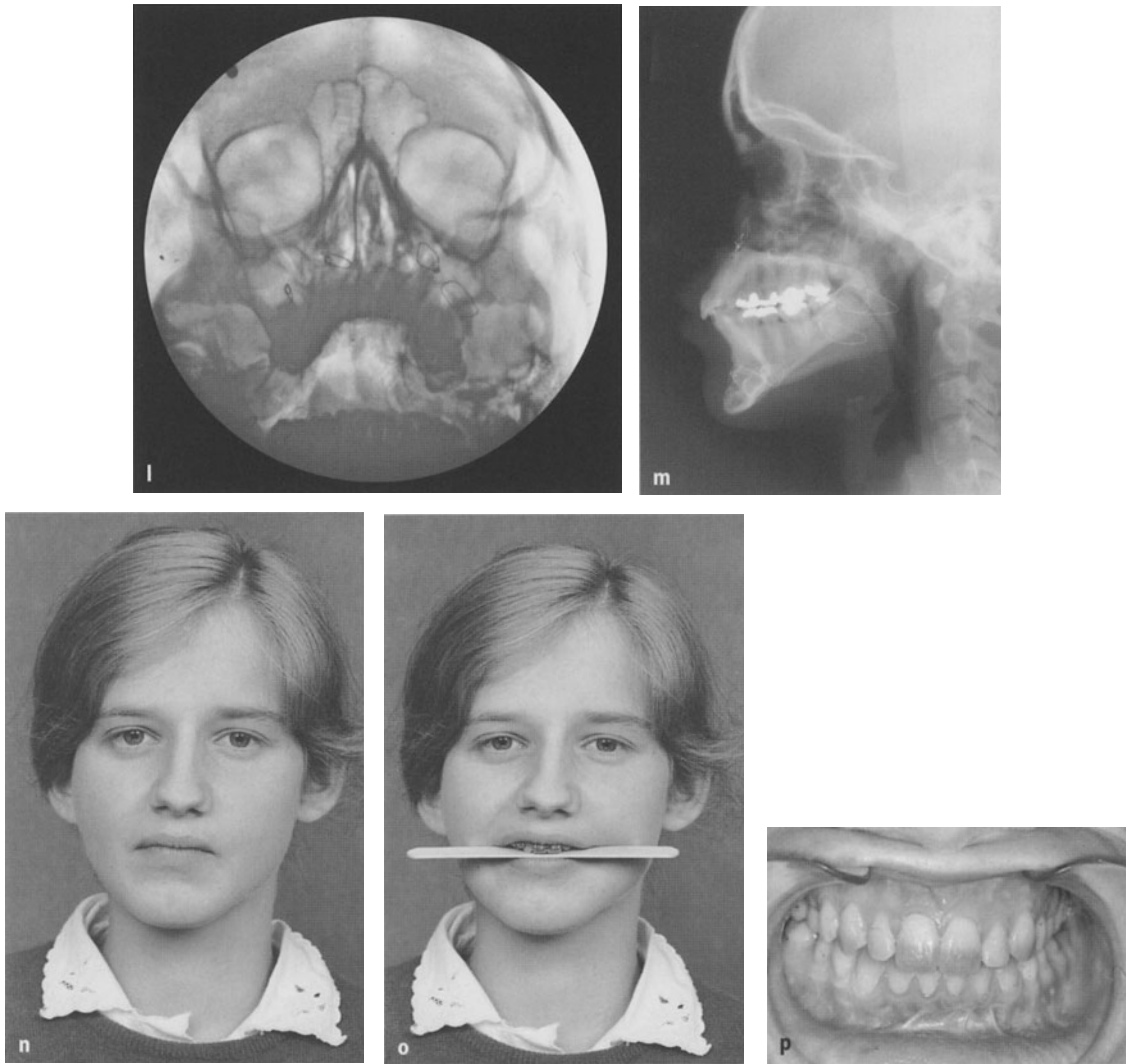


Fig. 24f–p. Unilateral hemimandibular hypoplasia after destruction of the condyle and ramus due to osteomyelitis in infancy, but with unhindered mouth opening. **f** The preoperative lateral cephalogram discloses the large discrepancy in body height between the two sides of the mandible and the remarkable shortness of the mandible. **h** Drawings of the plan for corrective surgery with rotation of lower half of the facial skeleton and reconstruction of the missing glenoid fossa and the ramus and the main part of the body by the use of rib grafts and additional chin symmetrization. **i** Mandible p.a. view 1 week after surgery demonstrates all surgical details and 2 years after the corrective surgery, the **j** panoramic view and the **k** mandible p.a. projection at maximum opening show the symmetrization of the mandible and maxilla achieved. **l** The Water's view, also 2 years after surgery, shows the result of the symmetrization of the height of the maxilla. **m** The lateral cephalogram, also 2 years after surgery, discloses the perfect skeletal framework for a pleasing profile line. **n, o** The patient's final front view, now with a horizontal occlusion plane. **p** The final occlusion with the result of the correction of the primarily contralateral rotation of the upper and lower rows of teeth

Actual Treatment and Result. The operation was performed according to the above plan with the use of three rib grafts, one of them as a costo-chondral graft. The rudimentary left condyle and finger-like left coronoid were removed. The surgery was executed by Dr. O. Hadjianghelou, then senior surgeon at my clinic. An excellent result was achieved from both the functional as well as the aesthetic points of view (Fig. 24i–p).

Discussion. Severe osteomyelitis of the condyle, ramus and posterior half of the body of the mandible during the first few months after birth had led to complete destruction of the growth capacity in these areas, resulting in a severe lack of growth of the mandible on the affected side with all its consequences, and yet with normal mouth opening. In retrospect it would have been wise to have replaced the infected parts of the mandible as ear-

ly as possible by a costo-chondral or iliac crest graft. We have done this very successfully in several tumour cases in early childhood, as well as in cases of destructive osteomyelitis of the mandible in adults by simultaneous resection and reconstruction via the oral route in spite of pus draining through fistulae, intra- and extraorally (see Fig. 29) (H. Obwegeser 1966b; H. Obwegeser and H. Sailer 1978).

Unilateral Hemimandibular Hypoplasia After Inflammatory and Traumatic Damage to One Condyle in Infancy (Fig. 25)

Sex and Age. Female, 17 years of age.

Aetiology and History. Recurrent bilateral otitis media, right side worse than the left, in infancy and early childhood. Mandibular asymmetry noticeable at 2 years of age. Torticollis on right side was operated at the age of 2. Patient was referred at the age of 17 to the Department of Orthodontics and Paediatric Dentistry of the Zürich Dental School (Head: Prof. Dr. Paul Stöckli, Ph.D.) because of a slight facial asymmetry and clicking in the right TMJ. I am grateful for his permission to use this diagnostically interesting case.

Clinical Findings. There is a slight facial asymmetry due to lack of vertical height on the right side, oblique occlusal plane and lip commissure, deviation of retruded chin to the right side (Fig. 25.a). On both sides there is a rather voluminous and strong masseter muscle, on the right side more so than on the left (Fig. 25b).

The occlusion shows deviation of the mandible to the right side by the full width of a lower incisor, with a slight crossbite on the right side (Fig. 25c). There was clicking in the right TMJ.

Radiographs. *Panoramic view at the age of 7 years 11 months* (Fig. 25) shows a rather voluminous mandible with a mixed dentition, with large rounded angles and a hypoplastic right condyle with a slim neck. The base of the maxilla is slightly higher on the right than on the left. The right ramus is slightly shorter than the left. Therefore there must be a slightly oblique occlusal plane. The left side of the mandible including ramus and condyle is within the normal range in structure. *Panoramic view at the age of 17 years and 4 months* (Fig. 25e) shows clearly hypoplasia of the right condyle and neck with a flat sclerotic surface and a rather small left condyle on a more than normally voluminous mandible, in particular in both angles and the left body regions. The right side shows a pronounced but shallow antegonial notch while the left horizontal ramus is quite a bit higher than average. There is a notch in the chin region. This seems to produce an asymmetry of the prom-

Conclusion. Severe osteomyelitis of the ascending ramus and condyle very early in childhood leads to an almost complete loss of growth capacity in spite of restoration of normal function. The “functional matrix” can obviously not stimulate growth of a sclerosed mandible and ramus.

inence of the chin to the left side, contrary to the clinical picture. There is a slight shortness of the right ascending ramus. The right side of the hard palate is lower than the left.

The cephalometric p.a. projection (Fig. 25f) at the same age shows clearly the hypoplasia of the right mandible and maxilla with deviation of the prominence of the chin and lower teeth to the hypoplastic right side.

The p.a. projection of the mandible in the open position (Fig. 25g) shows the pronounced asymmetry of the mandible and maxilla with shortness of the right ascending ramus with a small condyle and a medially tilted neck. The maxillary sinuses are unequal, the right is smaller than the left, and the floor of the nose as well as the maxilla is higher up on the right side than on the left. Mouth opening is very asymmetrical with deviation to the right.

Tomograms of TMJ areas taken at 17 years and 4 months: The right side showed a very small, partially sclerotic condyle with a short and thin neck; almost no glenoid fossa. The left condyle and glenoid fossa were also clearly smaller than normal for the age and size of the girl; the small condyle had very fine trabeculation with some minor sclerosis reaching down into the condylar neck and a “beak” on the anterior surface.

Lateral cephalometric view: Slight shortness of the mandible with a normal maxilla; different depth of the lower border of right and left mandibles; retrogenia of medium degree.

The result of *measuring the tracings of the panoramic radiographs* (Fig. 25h, i) at age 7 shows only a minor difference in length of the two ascending rami while that at age 17 discloses greater differences of the various sections of the mandible as one might have expected from the patient's appearance: the notch in the chin region does not coincide with the symphysis, as the Pg-point is easily recognizable on the panoramic view as a whitish spot, slightly to the left of the facial midline.

The right horizontal ramus is 14 mm shorter than the left. The right ascending ramus is also 14 mm short-

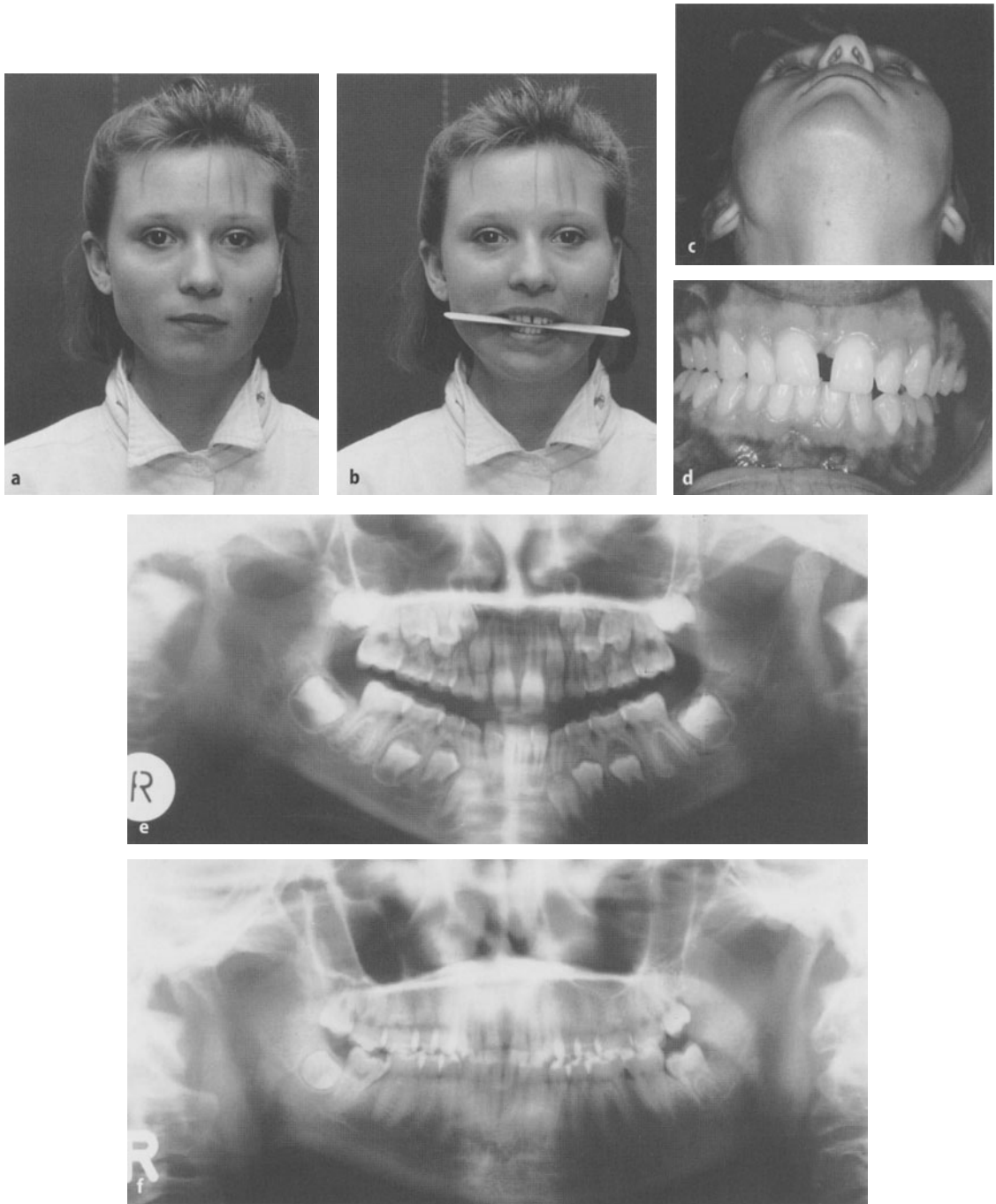
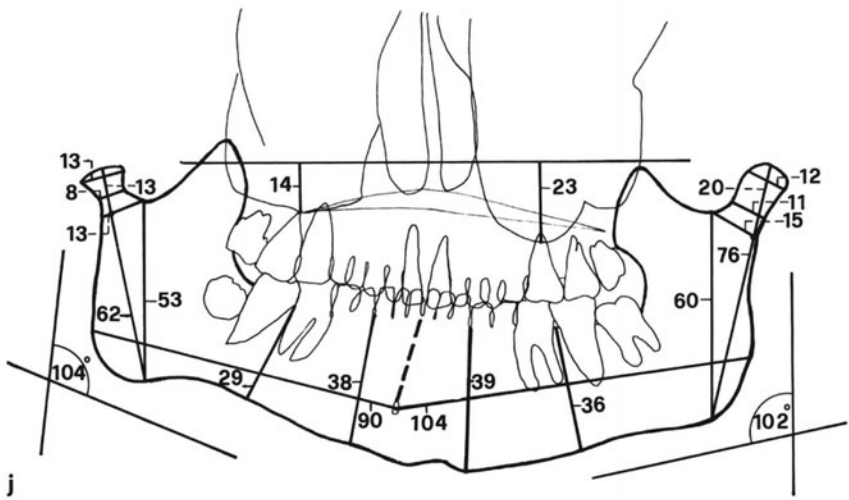
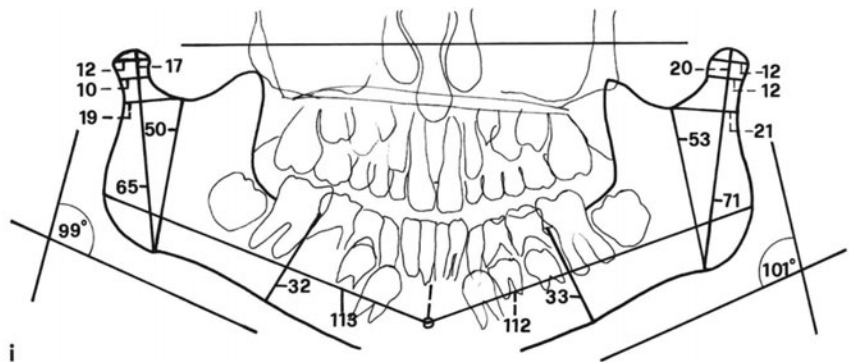


Fig. 25a–f. Case of unilateral hemimandibular hypoplasia after inflammatory and traumatic damage to one condyle in infancy. **a, b** Patient's front view with asymmetrical length of the two sides of the face, with an oblique lip commissure and chin deviation and an oblique occlusal plane. **c** Looking at the face from below discloses a clear deviation of the chin to the right side and a rather voluminous masseter region on both sides, the right more so than the left. **d** Patient's occlusion at the same age with deviation of the lower teeth midline to the right by the full width of a lower incisor and slight crossbite on the right side. **e, f** The panoramic radiograph at 7 years of age shows hypoplasia of the right articular process with a sclerotic surface of the too small condyle. At the age of 17 the patient resembles the typical appearance of a right condylar hypoactivity with a rather strong mandible of the bilateral masseter hypertrophy type



er than the left, as there is a 7 mm difference in the length of the trunks and 7 mm difference in the length of the articular processes.

Provisional Diagnosis. Hemimandibular hypoplasia on the right side, most probably due to condylar damage caused by infection of both joints in infancy (on right side more than on left), and additional trauma to the right condylar process. A medium degree of bilateral masseter hypertrophy.

Treatment Plan. The patient was twice informed in detail about the possibility of surgical correction of the asymmetry. A standard rotation procedure of the lower half of the facial skeleton including sliding genioplasty was proposed.

Actual Treatment. None, the patient refused any surgery.

Discussion. The asymmetry of the mandible due to the hypoplasia on the right side must have started very early in childhood. Because of the very hypoplastic and partially sclerotic right condyle and neck it is unlikely that it was of congenital origin. The repeated otitis media in early childhood must have led to the infection damaging both condyles, the right condyle more than the left. However, the medial tilting of the right condylar neck, seen clearly in the p.a. projection of the open mandible, could not have been caused by infection alone. It is more likely to have been caused by additional trauma in early childhood. As the left condyle is also slightly smaller than normal and has an anterior peak, that condyle too must have suffered because of the infection, but only a very little. The more severe damage to the condyle occurring in early childhood, probably at the age of 2 years, resulted in condylar hypoactivity on the more affected right side. This had led to hemimandibular hypoplasia on that side, but hardly recognizable because of the voluminous ramus-angle region.

The bilaterally present, rather voluminous and very rounded mandibular angles are typical features of the following possible origins:

1. Bilateral hemimandibular hyperplasia (it is not present in this case).
2. It may be seen in cases with a deep overbite. In this case this is not present.
3. It is seen in cases with a medium degree of masseter hypertrophy. This seems to be true for this case.

I have no explanation for the clearly inferiorly placed right side of the maxillary base. According to the shortness of the right ramus it should be the opposite. The only explanation I could think of is that it is a consequence of an earlier fracture of the maxilla. However, it is not recognizable in the panoramic view when the patient was 7 years old.

Conclusion. In early childhood, acquired condylar hypoactivity due to damage to the condyle caused by infection and trauma can be distinguished from congenital condylar hypoplasia, by three observations:

1. The shape and structure of the condyle in cases of bilateral congenital anlage-induced mandibular hypoplasia is almost normal, but its neck would be shorter and thicker, there would be no tilting of the neck nor any sclerosis of the condylar head and it would be equal on both sides.
2. Unilateral congenital hypoplasia of the condyle shows a small rather translucent condyle with no cortical covering.
3. The lack of downward growth of the maxilla and therefore the facial asymmetry is already noticeable at birth and not only at the age of 2–6 years (see case Fig. 16).

This case also demonstrates the necessity for radiographs in different projections. In this case, all other radiographs taken with the exception of the p.a. projection of the mandible in maximum opening did not disclose the medial tilting of the right condylar neck, which is definitely a sign of an earlier condylar fracture. Good TMJ–ramus tomograms might have disclosed it.

Fig. 25g–j. Case of unilateral hemimandibular hypoplasia after inflammatory and traumatic damage to one condyle in infancy. **g** The p.a. cephalogram at age 17 years and 4 months discloses a slight rotation of the lower half of the facial skeleton with a rather short right ramus. **h** The p.a. mandible at maximum opening, also at age 17 years and 4 months, shows a shortness of the right ramus and a clear bend of the right neck. Due to the shortness of the height of the maxilla on its right side and the severe deviation of the chin, the rotation of the lower half of the facial skeleton is even better recognizable. **i** The measurements on the tracing of the panoramic view at age 7 years show only a difference of 6 mm between the lengths of the ascending rami. **j** The measurements of the tracing of the panoramic view taken at age 17 years show substantial differences between left and right hemimandibles

10.2 Growth of the Mandible After Removal of Condyle?

10.2.1 Normal Growth of the Mandible After Removal of a Condyle Destroyed Traumatically Early in Childhood (Fig. 26)

Sex and Age. Female, 5 years of age.

Aetiology and History. One year previously, at the age of 4, the girl sustained facial trauma in a car accident. After the facial swelling had resolved a remarkably reduced mouth opening with deviation remained. A condylar fracture on the right side was diagnosed and treated with instrumental forced mouth opening. Because there was no improvement in mouth opening and because of the development of slight mandibular asymmetry the girl was brought for evaluation and possible treatment.

Clinical Findings. A charming girl 5 years and 4 months old, with slight asymmetry of the mandible in the closed position (Fig. 26a). The prominence of the chin is slightly out of the midline towards the right side. The right side of the mandible and the whole right side of the face is somewhat shorter than the normal rather flat left side. Maximum spontaneous mouth opening is 14 mm with some deviation to the fractured side. There is a perfect dentition according to her age and a perfect occlusion (Fig. 26b). Normal movements in the left TMJ, but only a little movement in the right TMJ area with a slightly hard prominence in front of the ear on palpation.

Radiographs. The panoramic view was not taken.

TMJ-ramus tomograms at maximum mouth opening (Fig. 26c,d) shows a normal, almost translucent condylar neck but with very little anterior movement on the left side. The left angle of the mandible seems normal and also the lower border in its posterior part. On the right side, the destroyed condyle is positioned lateral to the condylar neck. There is a slight but shallow antegonial notch. The ascending ramus height is reduced by the amount of the displacement of the condyle.

Provisional Diagnosis. Posttraumatic destruction of the right condylar head with fibrous ankylosis and slight asymmetry of the mandible with deviation of the prominence of the chin to the fractured side and reduction in the right facial height.

Treatment Plan. Removal of the damaged condyle and restoration of normal mouth opening, followed by postoperative mouth opening exercises with the help of dental appliances to secure symmetrical opening. Follow up for at least 12 months. As the girl was only 5 years old, one would normally not be sure that the necessary postoperative forced and checked mouth opening exercises could be guaranteed. However, her mother was a fully trained nurse. She agreed to supervise the necessary ex-



cercises and that the girl would accept the overnight wearing of a monobloc-like dental appliance.

Actual Treatment. Under endotracheal anaesthesia, the TMJ was approached via a retrotragal-temporal-incision. Lateral to the ramus-neck area there were several pieces of the condyle (Fig. 26e). They were removed and also the scar tissue in the joint region. A 46 mm mouth opening was achieved during surgery. A small pack of slices of lyophilized cartilage was fixed above the bone stump of the mandible. Fourteen days after surgery the spontaneous mouth opening was up to 35 mm. A dental appliance was constructed to be worn overnight and for 10 minutes every 2 hours during the day. Three months

after surgery, the child's mouth opening is normal (Fig. 26f). One year after surgery there is a 37 mm spontaneous mouth opening with little deviation to the operated side. There is still slight facial asymmetry at 4 and at 8 years after surgery. At age 15, with 45 mm mouth opening there is still a slightly noticeable facial asymmetry (Fig. 26g-i). Thirteen years after surgery, at the age of 18 there is practically no noticeable asymmetry of the facial contour, but a very minor almost unnoticeable asymmetry of the chin, perfect occlusion and spontaneous mouth opening of 46 mm with slight deviation to the right side. In spite of the lack of a proper right condyle, the young lady not only has a perfect occlusion (no orthodontic treatment was given), but also

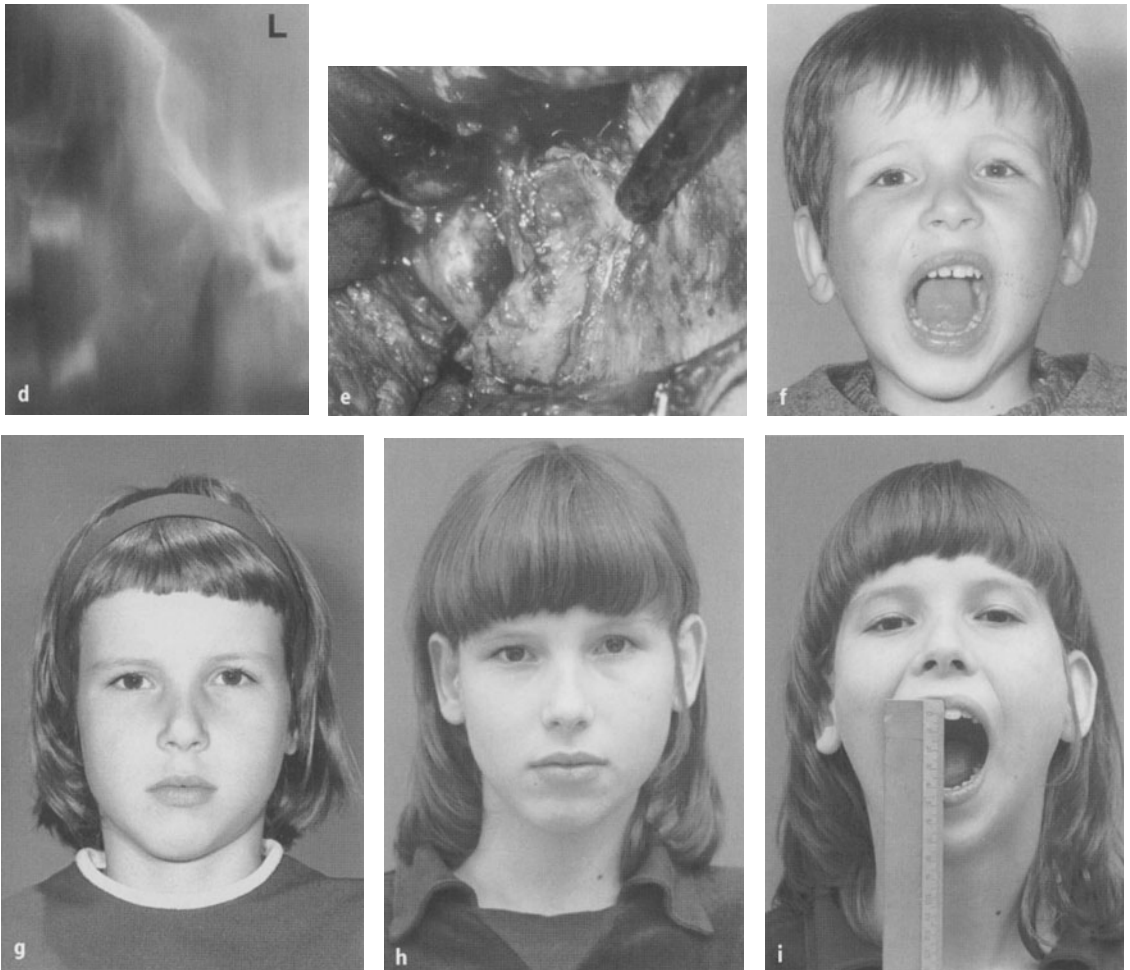


Fig. 26a-i. Normal growth of the mandible after removal of a traumatically destroyed condyle at 5 years of age. **a, b** One year after the accident there is slight facial asymmetry, but a good occlusion. **c, d** TMJ tomograms including rami: on the right side the condyle is destroyed and displaced and there is a pronounced but flat antegonial notch compared with the left side. The left ramus-condyle region is inconspicuous. **e** Operation view of the destroyed joint region: no normal anatomy, just a bony mass. **f** Very good mouth opening already exists 3 months after the release of the partial ankylosis and removal of all pieces of the destroyed condyle. **g** 4 years after surgery, some asymmetry of the mandible is still present and **h, i** 8 years after condylectomy the asymmetry had become less obvious. Mouth opening is 45 mm

almost normal forwards and lateral movement of the mandible (Fig. 26j–s).

The panoramic view, taken at age 18 (Fig. 26t) and the measurement of its tracing (Fig. 26u), discloses a normal mandible on the left side while the right shows a new condyle-like upper end. The right ramus is 10 mm shorter than the left while the difference in length of the trunk measurements is only 3 mm. This means that the main difference in ramus length is due to the difference in length of the two condylar processes. Although there is a pronounced antegonial notch, the length difference

between the two horizontal rami is zero. These measurements permit the conclusion that normal function after removal of the damaged condyle stimulates growth in length of both the horizontal as well as the ascending rami. These differences between the two mandibles do not show in the appearance of the young lady.

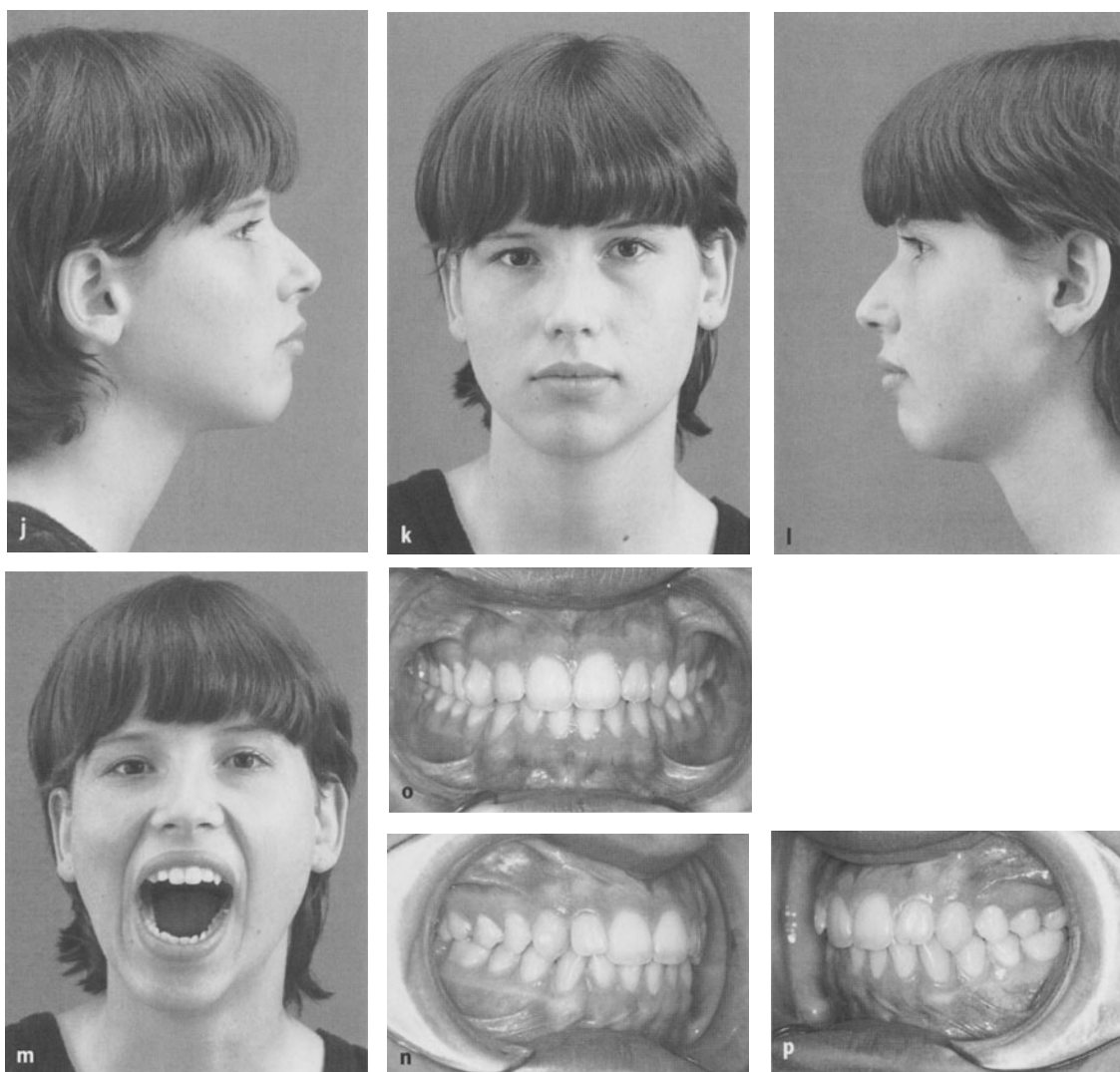


Fig. 26j–p. Normal growth of the mandible after removal of a traumatically destroyed condyle at 5 years of age. **j–m** At age 18 there is almost no asymmetry left except for a slight deviation on maximum mouth opening. No preauricular scar is visible because of the use of a retrotragal-temporal incision. **n–p** There is perfect occlusion and good movement of the mandible

Discussion. One year after a condylar fracture at the age of 4 years, with fibrous ankylosis and lateral dislocation of several pieces of the condyle, a clearly noticeable mandibular asymmetry had developed. The mandible developed absolutely normally after extirpation of the condylar remnants and establishment of a normal degree of mouth opening. There was no additional lack of growth or flatness on the affected side after normal function was restored: At the age of 18 there was a normal degree of mouth opening, a normal occlusion and normal mandibular movement except for a slight lateral excursion to the affected side in forward movement of the mandible. The consequences of the loss of the condyle are clearly visible in the panoramic view at age 18.

The abnormal shape of that half of the mandible cannot be overlooked. And yet the measurable differences are so small that the patient's appearance does not show them.

Conclusion. In a 5-year-old child, 1 year after a condylar fracture followed by fibrous ankylosis, a slight deviation of the chin and shortness of the mandible and facial height on the traumatized side is to be expected, even in centric occlusion. After removal of the traumatically damaged condyle and restoration of normal function there was no longer impeded growth of the mandible. One may not expect that the lack of growth, already existing at the time of the operation, will be restored with

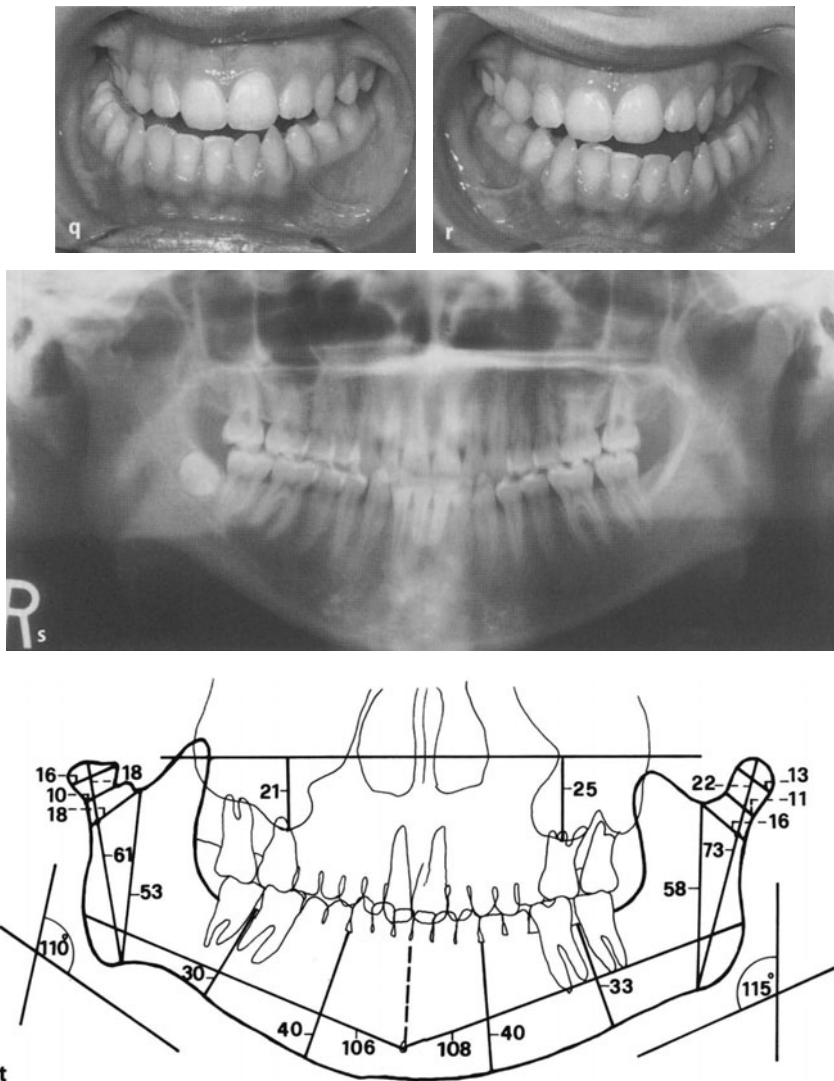


Fig. 26q–t. Normal growth of the mandible after removal of a traumatically destroyed condyle at 5 years of age. **q, r** The side movement of the mandible is within normal to both sides. **s, t** Panoramic view of the mandible at the age of 18 and the result of the measurements of its tracing

this treatment only. However, one dares to expect that the removal of the damaged condyle and restoring normal function will lead to almost normal development of the mandible, in shape, structure and function. This may suggest that any severely damaged condyle should be removed, even in early childhood, if perfect mouth

opening cannot be achieved. However, intensive postoperative treatment and care is absolutely necessary. This might not only be true for the traumatically damaged condyle but also for one which has been damaged by arthritis, infection or irradiation.

10.2.2

High Condylectomy Stops Hyperactivity Producing Excessive Hemimandibular Elongation with an Open Bite (Fig. 27)

Sex and Age. Female, 14 1/2 years of age.

Aetiology and History. Unknown. Nothing relevant in the family history. Commencement of open bite asymmetry 3 years previously at the age of about 11 1/2 years when the patient's face became longer. Referred by the Department of Orthodontics.

Clinical Findings. Looking at the patient from the front, the mandibular long face with chin asymmetry and a class III profile are very obvious (Fig. 27a). The chin deviates to the right side and so does the midline of the lower teeth by the width of half a lower incisor. There is a severe circular open bite, more open on the left than on the right side (Fig. 27b).

Radiographs. Panoramic view did not exist in those days.

The lateral views of the body and ramus revealed an elongated ascending ramus including the condyle and a rather stretched angle on the left side and a fairly normal configuration on the right side.

The mandible p.a. projection in the open mouth position (Fig. 27c) shows a severe mandibular asymmetry with elongation and stretching of the left side and deviation of the chin and of the lower teeth midline over to the right side. TMJ tomograms were not requested. Regrettably, all original radiographs from those days have been destroyed as mentioned in the introduction, however, the p.a. projection of the mandible still exists in rather poor quality, as a slide.

Provisional Diagnosis. Asymmetric open bite of unknown origin (in 1961).



Fig. 27a, b. High condylectomy stops hyperactivity producing excessive hemimandibular elongation with an open bite. **a** Front and **b** occlusal view of the girl at 14 1/2 years

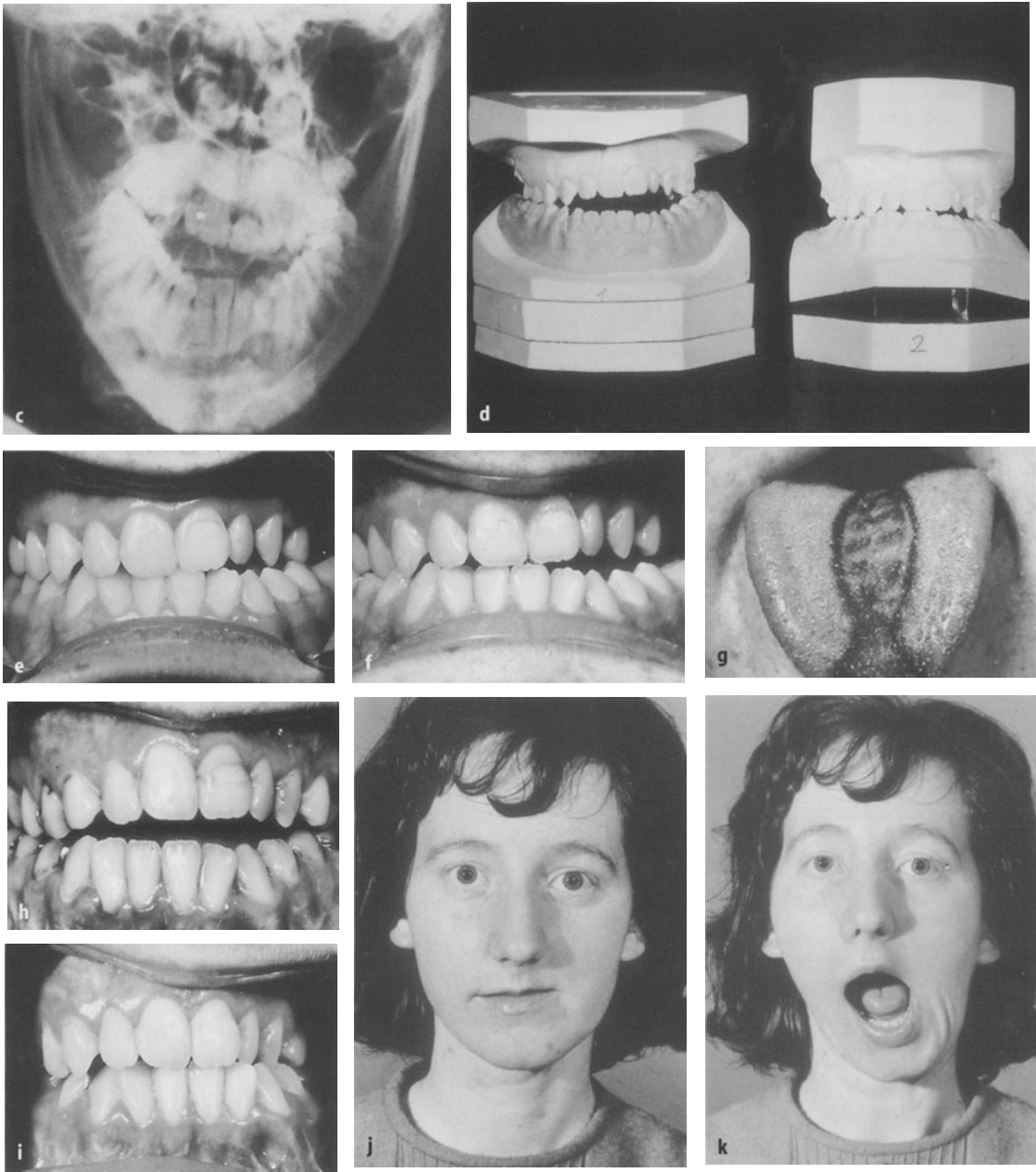


Fig. 27c-k. High condylectomy stops hyperactivity producing excessive hemimandibular elongation with an open bite. **c** The p.a. projection of the mandible in the closed position discloses a striking elongation of the left ascending ramus, producing an open bite. **d** According to the model-operation an acceptable intermaxillary relationship can be achieved by a bilateral sagittal splitting procedure. **e** Occlusal situation 8 weeks after a bilateral sagittal ramus splitting; now ready for orthodontics. **f** A recurrence of the asymmetrical open bite becomes obvious 11 months later. **g** The tongue was reduced in size, assumed to be a possible cause of the relapse. **h** The open bite and the asymmetry had increased quite a lot 1 year and 11 months later. **i-k** Left side full condylectomy resulted in the planned occlusion and a symmetrical facial appearance, but with severe deviation of the mandible on maximum opening

Treatment Plan. According to the model operation (Fig. 27d) an acceptable intermaxillary relationship could be achieved by a bilateral ramus sagittal splitting procedure. Postoperative orthodontic treatment should ensure a good occlusion. Presurgical orthodontics was refused.

Actual Treatment. At the age of 14 $\frac{1}{2}$ years, the planned intermaxillary relationship was achieved (Fig. 27e) using a bilateral ramus sagittal splitting procedure with circumferential wiring of the ascending rami, followed by 6 weeks intermaxillary fixation.

Eleven months later, it became obvious that the open bite was recurring and the midline of the lower arch of the teeth had shifted markedly more to the right side (Fig. 27f).

It was considered possible that the tongue might be the cause of the open bite (in those days). The author's type of tongue reduction (P. Egyedi and H. H. Obwegeser 1964) was performed (Fig. 27g). In spite of a much smaller tongue the open bite gradually increased. One year and 11 months after the tongue reduction, the open bite became unacceptable again and the mandible had shifted further to the right side (Fig. 27h).

It was now obvious that there was hyperactive growth activity on the left side. Because of this the whole left condyle was resected. Without any other additional surgery, the primary planned intermaxillary relationship and normal external appearance was achieved (Fig. 27i, j). Over several years there was no further tendency of the open bite to recur or for a mandibular shift to the right. Normal facial symmetry resulted. But on opening the mouth there was very severe deviation of the mandible to the side of the resection of the condyle (Fig. 27k). Postoperatively we had not used any guidance appliance for the mandible. After some years the patient suffered from severe TMJ arthrosis on the non-operated side.

Histology. An old low-power photomicrograph from the resected condyle displayed thickened articular tissue in the central aspect of the specimen. While the proliferative layer could not be identified in some places characterized by low cell density, the subjacent cartilage appeared similar to that seen in the H.H.-case shown in Fig. 36k. It resembled a transitional type of cartilage intermediate between hypertrophic growth cartilage and fibrocartilage, which disclosed a faintly visible deep layer of calcified cartilage. The almost continuous subchondral bone plate revealed only small marrow spaces frequently lacking direct contact with the cartilage. Thus, endochondral ossification was apparently minimal. Underneath the subchondral bone plate, the spon-

giosa exhibited large marrow spaces and few bone trabeculae that did not show any special orientation.

Diagnosis. Microscopically inconspicuous condyle lacking significant growth.

Comment: Unfortunately, there was only one photomicrograph available for examination. This slide did not disclose either signs of significant condylar growth or the structure of the subchondral spongiosa that seemed typical for other cases of H.E. (Fig. 79a). It would appear, therefore, that condylar growth had ceased by the time of surgery and, possibly as a consequence of growth termination, subchondral bone remodelled and assumed a normal appearance.

Discussion. In those days (1961) we were not aware that a condylar misregulation of growth existed. Then there was no possibility of proving condylar growth hyperactivity by radio-isotopes. No panoramic X-rays were available. It would have helped to have recognized early on that the left condylar neck, ramus and mandible were elongating so rapidly. Since we now recognize this type of pathophysiology we can, in retrospect, say that it was a very active H.E. In those days we also did not know that the tongue can never produce such a mandibular deformity. We now know that the tongue can only influence the position and axes of the teeth and the alveolar process but cannot change or influence the growth of the mandibular body or ascending ramus itself. We also know that complete condylectomy may result in severe deviation on opening the mandible, often with adverse consequences for the contralateral side.

The histological specimen of the resected condyle, regrettably, proved that abnormal growth activity did not exist at the time when the condyle was resected. This fact does not exclude condylar growth hyperactivity which must have been the cause of the abnormal growth in length of the same hemimandible. I am of the opinion that absence of a further relapse of the hemimandibular elongation with open bite after the condylectomy permits the conclusion that the cause of the abnormal growth had been within that condyle and no other cause was possible. Otherwise relapse would have occurred even after condylectomy.

Conclusion. In a case of severe condylar hyperactivity, the abnormal growth of the affected side is immediately arrested when a condylectomy is performed. Condylectomy on the affected side does not leave any chance for a relapse.

10.3

Growth Behaviour of Bone Grafts to Mandibular Defects

10.3.1

Normal Growth of an Iliac Crest Graft Replacing the Ascending Ramus with the Condyle and Half of the Body in Childhood (Fig. 28)

Sex and Age. Male, 10 years and 2 months of age.

Aetiology and History. Unknown. For the previous 6 months, a painless retromolar swelling which had increased in size had been noticed on the right side of the mandible.

Clinical Findings. A rather small boy, 10 years and 2 months old, in good general health with a slight swelling of the right mandibular angle region (Fig. 28a). Otherwise, extraorally nothing special. Motor and sensory functions of the facial soft tissues are normal. There is normal mouth opening. Orally there is a dense, painless swelling, the size of a small plum, in the right lower retromolar area. The overlying mucosa is adherent. It grows out of the mandible and is higher than the molar tooth in front of it (Fig. 28b). As a result of this, the teeth cannot be brought into occlusion without deviation of the mandible to the right side.

Radiographs. A panoramic view was not available.

Lateral view of mandible (Fig. 28c) and the mandible in p.a. projection shows a tumorous mass growing out of the right mandible in the angle region. There is no clear demarcation between tumor and normal bone. According to the report of the Radio-diagnostic Institute of the University Hospital it extends up to the semilunar notch and downwards to the mandibular canal.

Biopsy. A biopsy revealed the tumorous lesion to be a chondroblastoma.

Provisional Diagnosis. Chondroblastoma of right mandibular angle area extending upwards to the semilunar notch and forwards to the first molar.

Treatment Plan. Because of the histopathology diagnosis of "chondroblastoma" and the fact that there was no clear border between tumorous and normal bone tissue, disarticulation with resection just behind the mental foramen and possibly simultaneous reconstruction of the defect by an iliac crest graft was planned, using the oral route only, following our main principle: "*Any reconstructive surgery of the facial skeleton should be performed without incisions on the face*" (Principle Nr. 3).

Actual Treatment and Result. Under naso-tracheal intubation anaesthesia, the resection and reconstruction of the defect were performed via the oral route only. The resection included the overlying oral mucosa as well as the periosteum on the lingual and lateral aspects of the resected section of the mandible and the condylar process. The coronoid process was cut off and left in place. For the reconstruction, an iliac crest graft was taken and shaped according to the resected part of the mandible. It was inserted into the empty glenoid fossa and fixed to the decorticated lateral surface of the remaining mandible as an onlay, with one mattress wire suture.

Special care was taken in closing the soft tissues over the graft and adapting them to the graft with catgut threads passed around it, following the principle: "*Free bone grafts are like babies. They need to be kept warm by closely approximating the soft tissues around them*" (Principle Nr. 18).

The postoperative course was uneventful during 6 weeks of intermaxillary fixation and thereafter. The small 10½-year-old boy had a slight overcorrection of his right angle region (Fig. 28d). Normal mouth opening, function and occlusion were restored (Fig. 28e). The bone graft grew to be equal in all dimensions to the hemimandible on the other side during the period of normal skeletal growth. At the age of 22 years (Fig. 28f–l) there was no difference in length or in shape of the two sides. There was a normal occlusion and mouth opening without deviation and no facial scar because of the transoral approach.

Histology. The diagnosis of chondroblastoma was confirmed. No signs of malignancy were found.

Discussion. A non-vascularized bone graft from the iliac crest, replacing a resected large part of the right mandible including the ramus and condyle, can grow the same as the non-operated side. We have experienced this in several cases where a section of the mandible including the condyle had to be resected in early childhood. This shows that an iliac crest bone graft can, through function alone, produce length and mass of bone, just like a normal mandible with its condyle. So there is no need to introduce a growth centre such as a

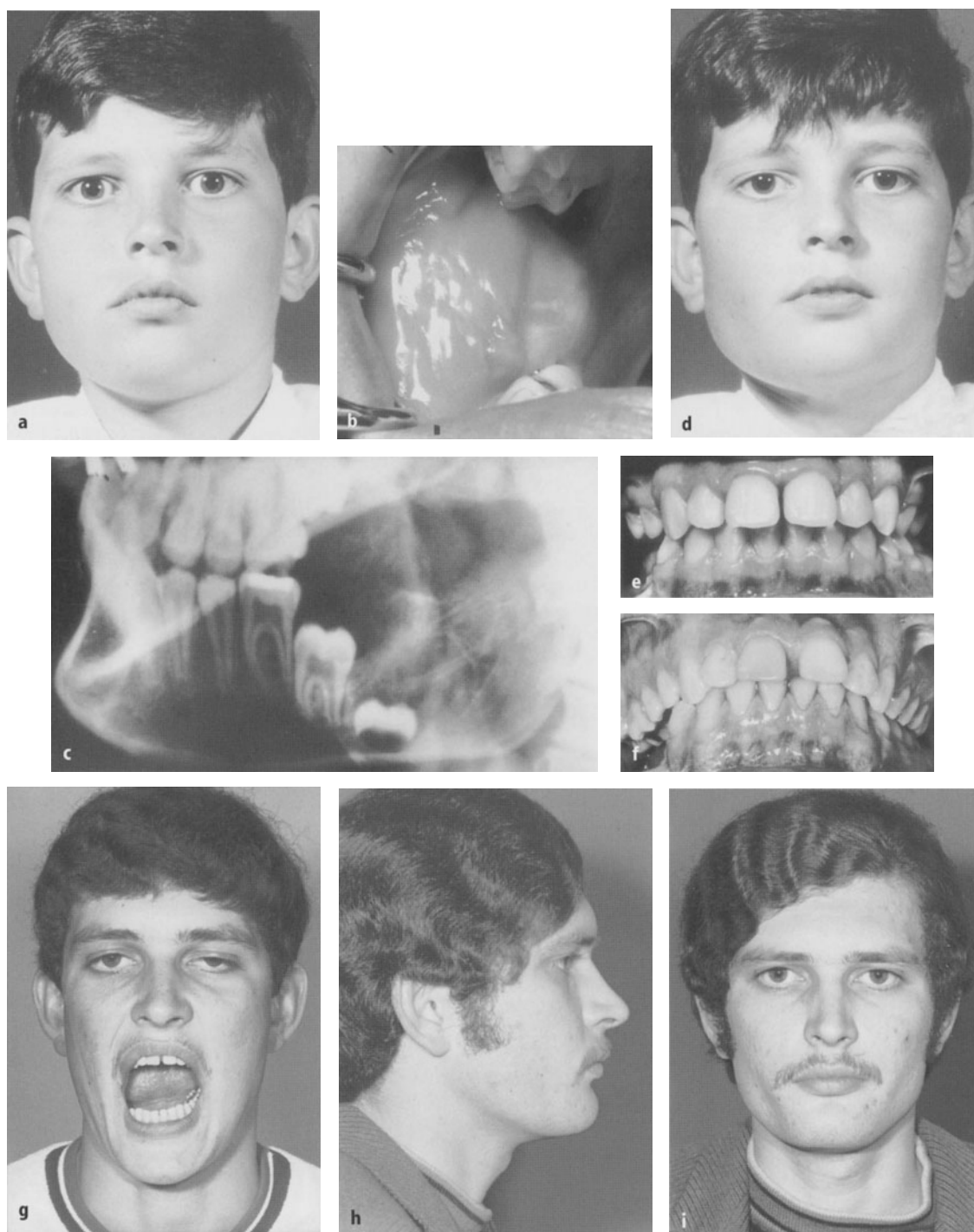


Fig. 28a-i. Normal growth of an iliac crest graft replacing the ascending ramus with the condyle and half of the body in childhood. **a, b** Appearance of boy of age 10 years and 2 months with diffuse swelling in the right angle region, also inside the mouth. **c** Oblique lateral projection of the right mandible discloses a tumorous growth without clear demarcation in the angle region of the 10-year-old boy. **d** There is overcorrection of contour of the right mandible 2 months after transoral resection of the ascending ramus including the condyle and half of the horizontal ramus and simultaneous reconstruction with an iliac crest graft. **e, f** The same occlusion 2 months after surgery and 12 years later, at age 22 years and 2 months. **g-i** Patient's normal appearance in front and profile views of the operated side, 12 years after surgery and normal mouth opening without deviation when growth had ceased

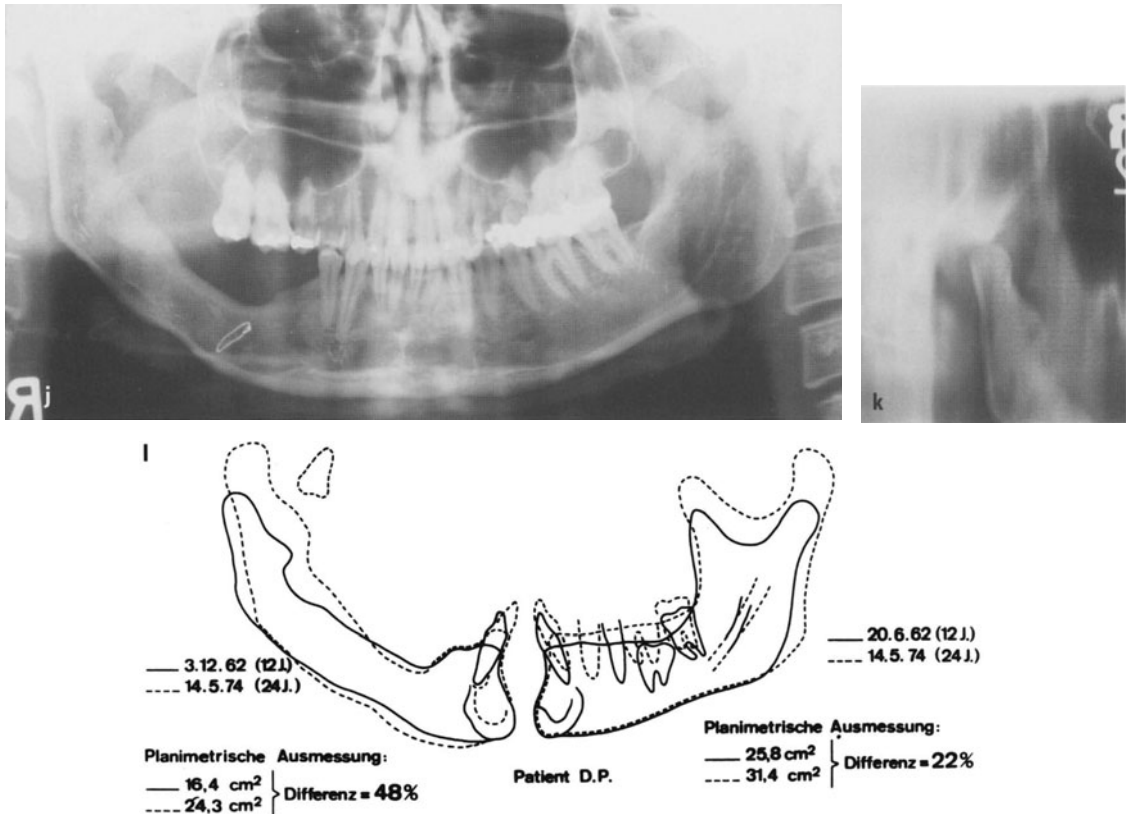


Fig. 28j–l. Normal growth of an iliac crest graft replacing the ascending ramus with the condyle and half of the body in childhood. **j, k** Radiographs taken 12 years after surgery: the iliac crest graft was contoured with a condyle-like end. It moves forward to the articular prominence, probably due to scar healing of the coronoid process to the iliac crest graft. **l** A comparison of the tracings of the two halves of the mandible 2 years and 12 years after surgery discloses clearly that the graft has grown more in length and volume than has the normal side of the mandible (by courtesy of H. Sailer)

metatarsal graft or a costo-chondral graft. An iliac crest graft is able to produce length and mass of bone, only by normal function. The growth is due to what is called the “functional matrix”. It must be the musculature. We do not know what the growth would have been if normal function had not been achieved. In cases in which a larger part of the horizontal ramus has to be included in the resection I prefer an iliac crest graft for the reconstruction rather than a costo-chondral graft. There are two main reasons for this: There is no overgrowth, but

the growth is equivalent to the other side and the iliac crest graft presents a perfect situation for later prosthetic work with or without implants.

Conclusion. A non-vascularized iliac crest graft replacing a large section of one side of the mandible including the condyle can grow to normal size, even in small children, when normal function is achieved. There is no other explanation for this growth other than the so-called “functional matrix”.

10.3.2 Growth Behaviour of Rib Grafts to the Mandible

Ribs are frequently used for the reconstruction of defects of the facial skeleton. I have used them as split rib grafts and also as full ribs.

For the split rib graft the full rib is widely decorticated, then split and then bent as needed by the use of a bone contouring forceps. I have often used single half ribs for orbital floor reconstruction following trauma, in craniofacial surgery, for the reconstruction of the hard palate in large clefts or defects after tumor resection. I have used them also with very gratifying results for the reconstruction of large skull defects (H. Obwegeser et al. 1978). Often, I have fixed a few of them together for the reconstruction of the orbits, in preprosthetic surgery for augmentation and for other various indications. According to my experience, the split rib grafts grow due to function, when inserted during overgrowth. But I have never seen a split rib graft produce overgrowth as if it had its own growth potential.

The full thickness rib graft has a much smaller field of indication, nevertheless it is a very important material in the reconstruction of large defects of the maxilla and the zygoma and even more so of the ascending ramus. For the latter indication it is mostly used as a costo-chondral graft, in adults and in particular in children. As the costo-chondral junction is known as the rib growth-steering centre, it is worthwhile to discuss its behaviour when used for reconstruction of defects of the mandible.

Through my own experience, as well as from reports in the literature, it is obvious that costo-chondral grafts can produce growth in length and in width (mass), at least in the same way as a growing rib behaves in the chest. But it can also behave differently. And what do we know about the behaviour of a full rib graft without the cartilage?

Growth Anomalies in Costo-chondral Grafts

Generally, one can expect the costo-chondral graft to develop in length equal to the normal side of the mandible. However, there are cases in which the graft may

lack sufficient growth. There are also two different growth anomalies known to develop in a costo-chondral graft to the mandible (B. MacIntosh and F. Henny 1977; B. MacIntosh 1985a, b, 1992).

1. Overgrowth in length

This often happens when the costo-chondral graft is inserted during the growth period (F. Pavel 1978; J. Munro et al. 1989; B. Guyuron and G. Lasa 1992; B. Svensson et al. 1993; N. Samman et al. 1995; A. Raustia et al. 1996; B. Svensson and R. Adell 1998). I have also experienced this (see Fig. 18).

2. Overgrowth in mass

Overgrowth in mass of the cartilage part of the costo-chondral graft has also been observed (J. Link et al. 1993; D. Perrot et al. 1994; A. Raustia et al. 1996). It is understandable that overgrowth in length can occur in a transplanted juvenile costo-chondral graft. It is more difficult to understand a tumour-like overgrowth like a chondroma, of its cartilaginous part. This abnormal growth behaviour of the rib cartilage is not known in thoracic surgery (W. Weder 1998, personal communication).

Astonishingly excessive tumour-like enlargement of the cartilage of the rib graft can even develop when the costo-chondral graft is transplanted into an adult. C. Politis et al. (1987) described this happening in the case of a woman who received the costo-chondral graft at the age of 22 and 2 years later, she became pregnant, without any problem on the grafted side. Then 2 years later, before the end of the next pregnancy, a tumour-like, excessive enlargement of the cartilage part of the graft occurred, with deviation of the mandible to the opposite side.

Overgrowth in mass of the bony part of a costo-chondral graft has not been reported. It could perhaps happen in a case of tumorous overgrowth of the cartilaginous part. If that enlargement is due to hyperactivity of the growth centre at the bone-cartilage junction. Then overproduction of bone of the rib graft could become understandable, as the growth centre is responsible for growth in length and thickness of the rib during the normal growth period.

General Enlargement of a Rib Graft Without Cartilage in the Adult (Fig. 29)

Sex and Age. Male, 45 years of age.

Aetiology and History. Five weeks before admission to our clinic, the patient had suffered a sudden severe pain in the left horizontal ramus accompanied by swelling of the soft tissues. After a few days, he went to see his general medical practitioner who gave him antibiotics. This did not resolve the situation. On the contrary, fistulae with purulent discharge appeared on the skin and intraorally along the left horizontal ramus. After 4½ weeks, the chin shifted over to the left side and the patient felt that his jaw had probably fractured ½ a week later he was admitted to hospital.

Clinical Findings. The patient was in very poor general condition (Fig. 29a, b). He was very pale and his laboratory findings proved the existence of severe infection. He had several fistulae discharging pus along his left horizontal ramus and also intraorally on top and on the buccal side of the edentulous left mandible. His chin was severely deviated to the left side which itself was shorter than the normal right horizontal ramus. There were clear signs of a fracture of the left horizontal ramus.

Radiographs. The lateral oblique view of the mandible shows the situation better than the panoramic view (Fig. 29c, d). The left premolar-molar region has been destroyed, showing osteolytic and sclerotic areas and

two rather large sequestrae, one of them very whitish. In that region there has been a fracture with a clear step and separation at the lower border, obviously due to a certain amount of upward rotation of the proximal fragment.

Provisional diagnosis. Destructive osteomyelitis with sequestration and spontaneous fracture of the left horizontal ramus.

Treatment Plan. One-stage operation for resection and reconstruction of the left horizontal ramus, from the parasymphiseal region to the angle of the jaw. Because of his rather poor general health a rib graft was to be used for reconstructing the defect, utilizing an oral access only.

Actual Treatment. Under naso-tracheal intubation anaesthesia 13 cm of the eighth rib, without cartilage, was removed from the left side. Its cortical covering was thinned, in a few areas of about 5–7 mm in length so much so that the bone marrow was clearly visible.

Next, the loose left second incisor was extracted and the mucosa and periosteum were incised on top of the ridge, through the fistulae, and all the way back, including half the height of the ascending ramus. The periosteum was adherent to the bone only in the chin and an-

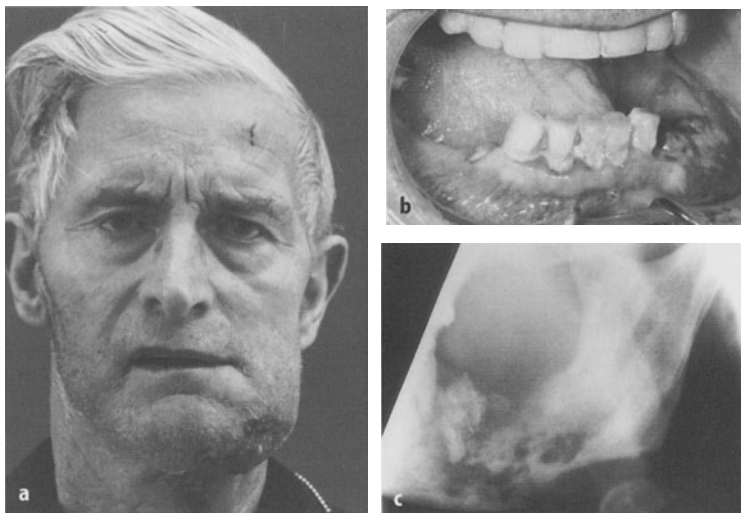


Fig. 29a–c. Enlargement of a rib graft used for immediate reconstruction of a horizontal ramus destroyed by osteomyelitis. **a, b** Patient's preoperative outward appearance and oral situation with several fistulae discharging pus. **c** Lateral oblique radiograph of the affected side of the mandible showing the extent of osteomyelitic destruction of the mandible and a large sequestrum better than the panoramic view

gle areas. The sequestrae were removed and the boundary with the non-infected bone was identified by making lateral cortical cuts, in the front as well as in the angle area until bleeding bone was encountered. I had learned that this is the best way to ascertain how far the osteomyelitis has extended (H. Obwegeser 1960). The stumps of the fractured mandible were resected back to sound bone. The periosteal tube, including the various

mucosal and skin fistulae, was carefully irrigated with an antibiotic solution.

Next, a continuous loop wire splint (H. Obwegeser 1952) was applied to the upper front and remaining four lower front teeth. Then the mandible was fixed to the maxilla. A horizontal groove was cut into the chin region, the width of the rib graft. At the angle, an additional piece of bone of the same diameter as the rib was cut

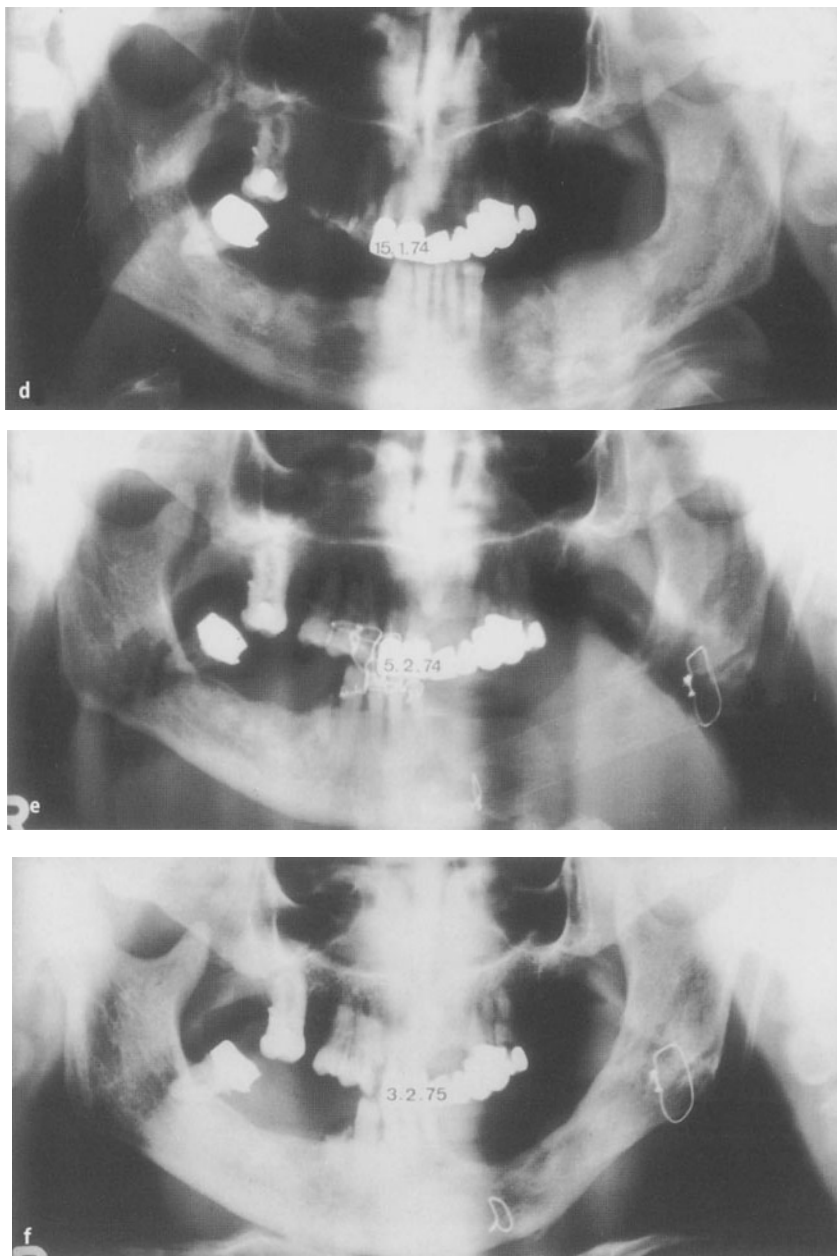


Fig. 29d–f. Enlargement of a rib graft used for immediate reconstruction of a horizontal ramus destroyed by osteomyelitis. **d–f** Panoramic view of the mandible before, soon after surgery and 1 year later

off at its inferior surface, leaving a finger-like 10 mm long spike of its upper part intact. A wire was placed like a mattress suture on each end. The rib graft was adjusted to the required length and size, placed into the prepared recipient bed and fixed to the stumps with the wire suture. Once more the operating field was irrigated with an antibiotic solution and the incision line carefully closed. Nothing was done to the various fistulae. There is no need to excise skin fistulae which develop from dental or bone pathology. They disappear spontaneously within a few days after elimination of their cause.

Postoperative Course. There were no postoperative problems. The patient received antibiotics for 8 days. The fistulae closed spontaneously within a few days. The intermaxillary fixation remained for 6 weeks. The rib graft was incorporated, without any abnormal movement. One year later, the initially rather thin and translucent rib graft had developed into a solid piece of bone, its structure on radiographs no longer diagnosable as a rib graft and the appearance was that of a normal mandibular body. It had also increased in its height and circumference (Fig. 29e, f). The patient's oral condition looked perfect as did his facial appearance (Fig. 29g, h).

Discussion and Conclusion. Overgrowth in length of a costo-chondral graft after transplantation in childhood seems almost a logical consequence of the fact that, in a child, the transplanted piece of rib would have had to el-

ongate more at its original location than a mandible would elongate at the same age. It is therefore no surprise that there are many reports in the literature of linear overgrowth of costo-chondral grafts after transplantation in childhood. It is rather astonishing that some will not grow enough. There is no reason and, for me, no explanation why in some cases the cartilage part will produce a tumour-like enlargement as this seems never to be the case in the chest after thoracic surgery (W. Weder 1999, personal communication). But, how can we explain that such an enlargement occurred in adulthood, during a second pregnancy after the graft transplantation (G. Politis et al. 1987). It is easy to say it was probably stimulated through the hormonal changes in pregnancy. That explanation one might accept if it had happened during the first pregnancy. Yet also then it was not possible to understand and prove it. So the only fact we can accept is that we do not know.

That the tumour-like enlargement of the cartilage part of the graft may push the mandible inferiorly and/or over to the other side is also understandable. But this does not produce a true H.H., although it may resemble other cases of tumorous or tumour-like enlargement of the condyle (see Fig. 38 and Fig. 39). The development of a true H.H., as a consequence of a costo-chondral graft, has not yet been reported in the literature and it would not be explainable from the pathophysiological view point. It would have to be proven not only radiographically but also by scintigraphy and histology.

While I normally prefer an iliac crest graft for reconstructing large defects of the horizontal ramus in chil-

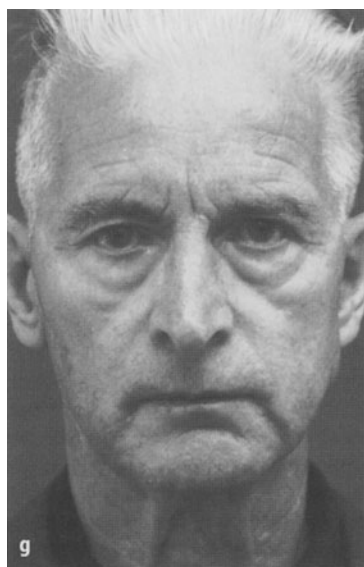


Fig. 29g, h. Enlargement of a rib graft used for immediate reconstruction of a horizontal ramus destroyed by osteomyelitis. **g, h** Patient's appearance and oral situation 1 year after resection of the osteomyelitic part of the mandible and simultaneous reconstruction using a rib graft without cartilage

dren as well as in adults, occasionally we used a rib graft, particularly in children with a hemifacial microsomia anomaly. The principal indication was the type of pathology necessitating resection of a part of the mandible and the patient's general condition, as harvesting a rib graft causes less morbidity than an iliac crest graft. We always carried out simultaneous resection and reconstruction with the same good results whether the operation site was infected or non-infected (H. Obwegeser 1968b). We know that an iliac crest graft will grow equal to the other side when inserted in childhood (see case Fig. 27) and we have learned that a rib graft without cartilage, used in an adult to replace a simultaneously resected part of the horizontal ramus, can also increase in volume.

The fact that a rib graft without cartilage replacing a diseased horizontal ramus will increase in volume can-

not be called a growth effect as it is the case with a costo-chondral graft. It is more likely due to function, in the same way as we have experienced with iliac crest grafts. When the graft is placed inside the periosteal tube then the increase in volume may also be due to that. We have learned that after subperiosteal resection of a large part of the horizontal ramus, spontaneous new bone formation can be expected when the periosteal tube is filled with some resorbable material, thus preventing a collapse of the tube. This has been demonstrated in animals as well as in man (G. Steinhard 1969).

One may experience routinely that a costo-chondral graft, transplanted for reconstruction of a large mandibular defect in early childhood, may not only grow in length but also in width and circumference to the same extent (see Fig. 18x,y) or even more than it does in the chest.

10.4 Deductions Drawn from These Clinical Cases

Hemimandibular Hypoplasia Without Condylar Hypoplasia

There are anlage-induced cases of symmetrical bilateral hemimandibular hypoplasia with retrogenia in which the condyles do not show any signs of hypoplasia, contrary to cases in which hemimandibular hypoplasia is the result of condylar hypoplasia due to either congenital or postnatally caused condylar hypoactivity. Also, contrary to the condylar hypoactivity cases, these cases have a short mandible with a rather voluminous shape.

In childhood, these cases seem ideal for correction by distraction osteogenesis. Correction in adult cases is performed according to the author's standard procedure (see case Fig. 11).

Bilateral Hemimandibular Hypoplasia Due to Postnatal Noxae

Noxious influences on the condyles in infancy or early childhood can lead to condylar hypoactivity with bilateral hemimandibular hypoplasia, producing symmetrical or asymmetrical micromandibulism. Such noxious influences may be juvenile (Still's disease) or purulent arthritis, untreated luxation of the condyles in infancy, a combination of juvenile arthritis and anlage-induced micromandibulism, irradiation damage and many others which affect the condyles.

In all these cases of micromandibulism due to damage to both condyles in infancy or early childhood, the amount of hypoplasia of both hemimandibles depends on the intensity and type of the noxious influence and the patient's age when it occurs (see cases Figs. 12–14).

The type of corrective surgery has to be chosen according to the patient's age. During the growing periods

it may be done by distraction lengthening of the ramus and also the mandibular body, or the transoral insertion of costo-chondral grafts on each side. In fully-grown patients, I suggest the use of procedures demonstrated in "Two ways to correct birdface deformity" by H. Obwegeser and O. Hadjiangelou in 1987 and in "Variations of a standard approach for the correction of the birdface deformity" (H. Obwegeser 1988). These procedures are, either a vertical osteotomy in the mandibular body on each side with insertion of a block of iliac crest bone, or the following procedure is performed in one-stage: extraction of at least one bicuspid on each side and surgical repositioning of the anterior teeth segment for closure of the gaps and for correction of the anterior angulation of the axes of the anterior teeth into normal position. Then the mandible is elongated by a bilateral, long surface, sagittal splitting procedure and a double chin-slide advancement.

Normal Development of a Hemimandible in Spite of Congenital Aplasia or Hypoplasia of the Condyle

Congenital aplasia of the condyle can still permit fairly normal development of the affected side of the mandible. But the lack of length of the whole ascending ramus, because of its absent condyle and condylar neck, produces a very pronounced clinical picture of severe congenital or in-infancy-acquired condylar hypoplasia with deviation of the mandible, disturbed occlusion and a severely oblique occlusal plane.

Early reconstruction of the absent condyle or replacement of the hypoplastic condyle with a costo-chondral graft can avoid the skeletal consequences of hemimandibular hypoplasia (see case Fig. 15).

Congenital or Acquired Condylar Hypoactivity?

In cases of condylar hypoplasia, it is sometimes difficult to diagnose whether the case is an unilateral congenital condylar hypoactivity or one that has been acquired early in life. The acquired type should mostly be diagnosable from the case history and by the shape of the condyle as viewed on standard radiographs and CT-tomograms. The congenital form may not be diagnosed on the radiographs, as they can show, in mild forms, condyles of almost equal size. However, since the condylar hypoactivity must have already been present during embryonic growth, such a case will show very early an oblique lip commissure with the corner of the affected side higher than the opposite side. There should also be a slightly oblique occlusal plane (see case Fig. 16), when the hypoactivity is unilateral.

Treatment should depend on the degree of asymmetry and the patient's age.

Hemimandibular Hypoplasia or Contralateral Elongation?

In some cases it is not easy to diagnose whether a case is an unilateral hemimandibular hypoplasia or a contralateral normal or even hyperactivity case as seen in hemimandibular elongation. This is particularly true in childhood during the growth period. Although there may already be a noticeable asymmetry, the radiographs may still not show clearly a difference in size between the left and right sides. Scintigraphy can also be inconclusive during growth. Even if there were an intensity difference in cellular activity in the condylar areas, one can still interpret the one side as hypoactive or the normal side as hyperactive. In these doubtful cases the deformity can be very mild (see case Fig. 16) or so severe (see case Fig. 69) that something has to be done immediately. Study of the case's occlusion, the midline of the lower teeth and chin and the radiographs will not always guarantee the correct diagnosis. In hemimandibular hypoplasia the midline of the lower teeth never moves over to the other side (see case Fig. 16) but to the side of the hypoplasia.

The treatment plan must be: in mild cases wait, observe and perform the final correction when the diagnosis is clear or when growth has finished. Presurgical orthodontics is usually required. In severe cases (see case Fig. 28), the overgrowth has to be stopped surgically by a high condylectomy, or the growth of the hypoplastic side increased by a costo-chondral graft. As long as growth has not ceased, a distraction osteogenesis procedure may not produce a complete correction but can result in considerable improvement.

Differential Diagnosis Between Hemimandibular Hypoplasia and Contralateral H.E.

Some cases of hemimandibular hypoplasia enable a clear diagnosis to be made, but the aetiology may remain unclear.

When there is an oblique lip commissure with one corner further lateral and higher than the other, it is very obvious that that side is hypoactive, consistent with hemimandibular hypoplasia. H. E. does not produce a higher corner of the lip, nor does it push it inferiorly, so it can not be a contralateral H.E. A contralateral H.H. would lower its lip corner. But a contralateral H.H. would be easily diagnosable radiologically.

Management will consist primarily of clear information to the patient's parents and operation at the request of the patient, at some future time.

Hemimandibular Hypoplasia in Hemifacial Microsomia Cases Is Different

The hemimandibular hypo- or partial aplasia in cases with unilateral hemifacial microsomia differs in its aetiology from those with congenital or acquired condylar hypoactivity. Also its clinical picture is very different (see cases of Figs. 17–19). It is due to intrauterine damage of various degrees to skeletal parts, mainly the mandible, as well as the surrounding soft tissues. An existing hypoplastic condyle in these cases in a 1½-year-old girl showed a much-too-thin hypertrophic cartilage layer in relation to the size of the very small condyle (see case Fig. 19).

The treatment for these cases, in infancy or early childhood, is the reconstruction of the missing glenoid fossa and hypoplastic or missing ramus with a costo-chondral graft (see case Figs. 18, 19). In cases after growth has ceased, correction will be carried out according to the procedure published by the author in 1970 and 1974. It consists of the rotation of the lower half of the facial skeleton and the reconstruction of the missing skeletal and soft tissue parts with rib and/or iliac crest grafts, which is followed 6 months later by non-anastomosed free fat transplantation and onlays of slices of lyophilized human cartilage for final bone and soft tissue contour corrections. In order to avoid visible facial scars the skeletal reconstruction is performed transorally and, where necessary, from an additional temporal or retrotragal approach. The fat is also inserted via a retrotragal-temporal incision or from an incision behind the ear (see case Fig. 17).

Hemimandibular Hypoplasia – A Serious HAZARD WARNING!

There are rare cases of hemimandibular hypoplasia which show a similar skeletal abnormality as does a medium-degree hemifacial microsomia, even including an oblique occlusal plane. As in other cases with a similar appearance, the surgeon may be inclined to correct the skeletal asymmetry by a rotation of the lower half of the facial skeleton and run into a disastrous surgical situation because of torrential bleeding due to a perimandibular and perimaxillary cavernous haemangioma. Consequently one must try to eliminate diagnostically the existence of such a cause of the skeletal anomaly before any surgery is performed. There is still the possibility of surgical correction of the asymmetry in such cases (see case Fig. 20).

Non-damaged Condyle Provides Normal Growth of the Mandible on That Side in Spite of Lack of Function

A posttraumatic TMJ ankylosis acquired at the age of $1\frac{1}{2}$ years, which does not permit any degree of mouth opening leads by the end of the growth period to a severe lack of growth of the mandibular half on the affected side. But the contralateral half grows normally in spite of lack of function. So, function is not necessary for the development and growth of a hemimandible as long as its condyle is normal and not damaged (see case Fig. 22).

The treatment after cessation of growth is more difficult than in cases with a symmetrical birdface. Therefore, I first make out of the asymmetrical birdface a symmetrical one, and then I handle the case in the same operation in the same way as a symmetrical birdface. This is not the only possible way of handling an unilateral birdface appearance. By elongation of the short side with a bone graft within the body excellent results can also be achieved (L. Merville 1970; H. Obwegeser and O. Hadjiangelou 1987).

Does a Damaged Condyle Prevent Normal Mandibular Growth?

If a TMJ ankylosis, acquired in infancy after purulent otitis media, is successfully released in early childhood, resulting in good mouth opening, the affected side of the mandible may still end up with a much too short ramus and mandibular body, in spite of restored function. This occurs when the damaged condyle is not completely removed during the surgery to release the ankylosis. So, function cannot overcome the negative influence on growth of a coexistent damaged condyle (see cases of Figs. 23 and 24).

The correction in childhood may be achieved by a bone distraction procedure via the oral route, or a costo-chondral graft. For corrective surgery in adult cases I use the same procedure as demonstrated in case Fig. 22.

Osteomyelitis of Ramus and Condyle in Infancy Produces a Picture Similar to Hemifacial Microsomia

Osteomyelitic destruction of the condyle and ramus in early infancy may lead to an almost complete loss of the affected structures, resulting in an extreme picture of hemimandibular hypoplasia. The skeletal picture is almost equal to that seen in severe hemifacial microsomia (see case Fig. 24).

Treatment should include early reconstruction of the ramus with a costo-chondral graft inserted into a reconstructed glenoid fossa. After cessation of growth the treatment is almost the same as the reconstruction of the facial skeletal abnormality in cases of rather severe hemifacial microsomia.

Radiographs in Different Projections Are Often Essential for Proper Diagnosis

In hemimandibular hypoplasia, an inflammatory TMJ episode in infancy may probably have been the cause of the condylar hypoactivity. But maybe additional trauma to the condyle can influence the condylar growth activity. Case Fig. 25 demonstrates the necessity for standardized routine radiographs in every case.

The treatment depends on the patient's wish to have the asymmetry corrected or not. Surgically, the treatment does not pose any problems in such a mild case of hemimandibular hypoplasia.

Remove the Traumatically Damaged Condyle in Cases of Ankylosis!

The removal of a traumatically damaged and dislocated condyle in early childhood is not going to do any harm to the further growth of that hemimandible (see case Fig. 26). On the contrary, it permits normal further growth in an already slightly hypoplastic hemimandible when good function is achieved. This fact suggests that a damaged condyle can act as a brake on growth.

In several cases I have observed normal growth of the affected hemimandible after release of a TMJ-ankylosis in early childhood, in both unilateral as well as bilateral cases. Besides the removal of the damaged condyles intensive postoperative function is necessary. For that, supervised and assisted postoperative exercises are mandatory for at least 1 year. However, the already existing growth deficit will not be corrected by the further growth.

Function Can Allow an Iliac Crest Graft to Grow

In childhood, function seems to encourage an iliac crest graft to grow in length equal to the other side of the mandible, when it is used for reconstruction of a large section of a hemimandible including the condyle. Yet an iliac crest graft does not contain any growth centre. This means that an iliac crest graft receives growth stimulation from function only. So, in order to have a piece of bone growing in length which does not have a growth capacity of its own, the “functional matrix” can make it grow (see case Fig. 27). We have experienced this in 4 cases (H. Sailer 1977).

The Condyle Is Responsible

High condylectomy prevents relapse in a case of severe H.E. This is a very important fact. It proves that the steering agent for the hyperactivity must be within the high condylar layer, and means that a regulator responsible for that additional in growth length must exist. If that is the case, then there must also be place for a regulator responsible for production in mass of the same hemimandible. The conclusion is that two growth regulators must exist within the high condylar area (see case Fig. 28).

We know through several cases that further growth of the mandible is not interfered with in spite of a high condylectomy.

A Damaged Condyle Is the Worst Thing for Normal Mandibular Development

On the basis of experience with the cases described, it seems that the worst thing for normal growth of the mandible is a damaged condyle, whether it is of embryogenetic developmental origin or it has any postnatal cause. Since normal growth occurs after removal of a traumatically destroyed condyle and even an iliac crest graft produces growth equal to the other side when normal function is achieved, one might suggest that at any stage of growth a damaged condyle is better removed than left in place, as long as normal function can be achieved postoperatively. I therefore also suggest high condylectomy in juvenile arthritis cases because we know that damaged condyles will not be able to produce normal mandibular growth but may act as a brake on growth.

This philosophy does not mean that in every case of condylar fracture the condyle should be removed when growth without normal function can be achieved. I am a strong supporter of non-surgical conservative functional therapy of condylar fractures whenever possible. Already long ago, large statistics (H. Köle 1955; O. Herfert

1955) and our experience tell us that normal mandibular growth can develop, in juvenile and even in early infancy cases of condylar fractures, by conservative functional treatment. But when such a degree of lack of mouth opening results that surgical release becomes necessary, then the condyle should not be left in situ.

Costo-chondral Graft for TMJ Reconstruction

The costo-chondral graft is an ideal material for reconstructing a ramus-condyle defect, in adults as well as in children. This is especially true in cases of hemifacial microsomia. It will normally grow equal to the contralateral side, but it can also lack sufficient growth, overgrow in length or produce a tumour-like enlargement of the cartilage part causing a shifting of the mandible to the contralateral side.

A rib graft without the cartilage is a very useful substitute for mandibular defects in cases where general health problems preclude harvesting sufficient bone from other sites. When the rib graft is placed into the periosteal tube it can increase in volume remarkably; also pre-existing severe infection is no contraindication to its use for defect reconstruction.

What I know About the Condyle, Derived from These Cases

1. The condyle is necessary for normal function of the mandible. But even without a condyle the mandible can function satisfactorily with proper training and physiotherapy after the condyle removal (see cases of Figs. 26 and 27).
2. The undamaged condyle ensures normal growth of the mandible in spite of lack of function (see case Fig. 22).
3. Normal size and shape of the condyle may not guarantee normal growth of the mandible in spite of the existence of normal function (see case of Fig. 11).
4. When the condyle is damaged, the mandible may not develop to a normal shape and size in spite of normal function being present (see case of Figs. 12–25).
5. The condyle is not necessary for normal development of the mandible. When the condyle is missing, the functional matrix can take over and result in a mandible normal in size and shape (see cases of Figs. 15, 26 and 27). But the functional matrix cannot overrule the negative growth influence of an existing damaged condyle.
6. Only the condyle can develop a specific hyperactive influence on the growth of the mandible of the same side (see case Fig. 28). Without a condyle H.H. and H.E. do not seem possible.

Condylar Hyperactivity

Condylar Hyperactivity

11.1

Introduction

In contrast to the discussed congenital or acquired unilateral or bilateral forms of hemimandibular asymmetry due to condylar hypoactivity the aetiology of which is known, there are postnatal, mostly asymmetrical, unilaterally-developing forms of anomalies of the mandible for which we do not know the cause.

They are neither congenital nor are they hereditary or anlage-induced. They develop without a known causation during the course of growth or even later; and they always present themselves with the same typical appearance, but in gradual gradations.

They are mandibular asymmetries which are due to a surplus of growth activity which affects the whole hemimandible as far forward as the symphysis and no further. They are very typical form anomalies which develop only during the growth period or even later independently of sex and side and they occur in various grades. They never seem to exist as congenital forms. They can develop in rudimentary form only, in clear classical form, or excessively. They can appear very slowly over several years or very rapidly within a few weeks or months. They may become apparent in early childhood by the age of 6–7 years and occasionally earlier, or at any time during the later growth period. Rather rarely, they can start even after general growth has ceased. They can appear in an unilateral isolated form, or as a hybrid and they can appear also asymmetrically bilaterally.

These asymmetries do not depend aetiologically on noxious stimuli. At least never in these forms has a noxious cause been recognized, such as the aetiological causes mentioned in the list “Unilateral condylar hypoactivity due to condylar damage by adverse factors in early childhood”. They are the product of misregulation of growth of unknown origin. If such anomalies seem to appear bilaterally symmetrical then they are not classifiable as cases of misregulation of growth. Such cases belong to the group of anlage-induced mandibular anomalies, like ante- and retromandibulism, except when they are caused by a central steering mechanism as seen in acromegaly.

Depending on the intensity of hyperactivity and the patient's age when the abnormal growth begins, the opposite side and the maxilla may also be influenced in

their development and I am of the opinion that they are caused by hyperactivity of two different growth regulators. The more rare form is characterized by an increase in volume of all parts of the affected side of the mandible, without pushing the prominence of the chin over to the opposite side. It ends exactly at the symphysis. The more common form is characterized by elongation of all parts of the affected side mostly with widening of the gonial angle – often without elongation of the condylar neck – and a clear displacement of the prominence of the chin and the midline of the lower incisors to the opposite side. However, the elongation also stops exactly at the symphysis. Occasionally there are hybrid forms of these two anomaly types, whereby one or the other type can be more dominant. We have called these two anomalies Hemimandibular Hyperplasia (H.H.) and Hemimandibular Elongation (H.E.) (H. Obwegeser and M. Makek 1986a,b).

Based on the clinical appearance and on experience in dealing with them, we are convinced that the steering mechanism for the growth of these anomalies must be located entirely within the cartilaginous zone of the condylar head. This hypothesis is proven by the fact that in all very rapidly developing cases, the abnormal growth has immediately ceased after a high condylar resection has been performed.

11.2

Three Types of Condylar Hyperactivity

There are three classical variations of condylar growth hyperactivity which are clinically distinguishable without any radiographs. It seems as if our primary publication on this subject (H. Obwegeser and M. Makek 1986a,b) was not clear enough regarding classification. Quite a few authors mention in their recent publications that we distinguished two different types of condylar hyperplasia as do some other authors, using other terms. I want to clarify that I do not talk about different types of condylar hyperplasia. I prefer the expression condylar hyperactivity. There are three clearly definable forms of condylar hyperactivity and only two of them produce condylar hyperplasia: the pure H.H. type and the hybrid form of H.H. and H.E. The pure H.E. type never produces condylar hyperplasia.

Type I

This form presents with surplus growth in volume as its outstanding feature. The mandible on the affected side becomes larger in volume in all its sections, the condyle and its neck, the ascending ramus and the body, but it extends to the symphysis only, without shifting the chin prominence to the contralateral side. The occlusion is somewhat rotated. But there is no crossbite. This is the classical picture which I have called “hemimandibular hyperplasia” (H.H.) (see Fig. 1a).

Type II

This is the most common. There is elongation of one half of the mandible with displacement of the chin prominence to the other side and with a typical crossbite, but without an increase in volume of the affected side. Normally the elongation is located in one hemimandible only and it always stops at the symphysis. I have called this type of mandibular asymmetry “hemimandibular elongation” (H.E.) (see Fig. 1b).

The elongation is not limited to the horizontal ramus only, but it is an elongation of all sections of that mandibular half, the horizontal and the ascending rami and the condylar neck and head. We distinguish two varieties, a slender and a non-slender type. They differ in shape and amount of elongation.

These two growth anomalies, Type I and II, differ completely in their appearance when they are in their pure form. Therefore, I am of the opinion that they are caused by hyperactivity of two different growth regulators.

Type III

The third form develops through elongation and hyperplasia on the same side. In such a case the affected side will not only be more voluminous with its lower border further inferior than the other side, but the chin prominence will also be shifted over to the other side and the occlusion will be rotated. In gross cases the dental arch of the mandible is rotated and out of occlusion. It is a combination of the occlusal situation found in pure cases of H.H. and H.E. This third form is a hybrid form of H.E. and H.H. and I call it the hybrid or mixed form of H.H./H.E. It can produce the most grotesque types of mandibular and facial asymmetry (see Fig. 1c).

11.3**Nomenclature for Condylar Hyperactivity Forms**

All three forms are generally subsumed in the literature under the term “condylar hyperplasia” (M. Rushton 1944; N. Rowe 1960; P. Cernea 1967; R. Walker 1967; R. Bruce and J. Hayward 1968; J. de Burgh Norman and

J. Painter 1980; among others). Some authors distinguish different forms, but without clarifying their distinctive features precisely, neither in description nor by radiography. The expressions laterognathia or unilateral or deviation prognathism is used, probably for the unilateral H.E. (R. Bruce and J. Hayward 1968; P. Mercier 1969; and many others). L. Kaban (1988, 1990) distinguishes between a vertical and a rotational pattern of condylar hyperplasia. J. Delaire et al. (1983) and A. Gondeeff et al. (1988) speak about a L’hypercondylie mandibulaire unilatérale ou bilatérale à croissance verticale et transversale. J. Hovell in 1963 has already described the three different forms under the term condylar hyperplasia. He did not give each of them a distinct name but otherwise he described exactly the same three types as I have done. But he had no explanation for their cause.

11.4**Forms of the Condyles**

As all three types of condylar growth hyperactivity, in the literature, are generally subsumed under the term “condylar hyperplasia” one would have to expect that all these three forms produce a hyperplastic condyle. This however, is not true. It is only valid for the H.H. types and the hybrid types. Without a hyperplastic condyle such a growth anomaly does not belong to one of these two groups. It is not valid for the H.E. type.

The enlargement of the condyle can be very mild and it may be of almost normal shape. Most will have an anterior “beak”. It can also be a very irregular enlargement of the condyle (P. Cernea 1967; R. Walker 1967; R. Bruce and J. Hayward 1968; P. Egyedi 1969; J. de Burgh Norman and J. Painter 1980; among others). Several authors have shown this in drawings.

Nowadays, one can obtain a clear picture of the shape of the condyles without removing them. Three-dimensional computer tomography and also stereo-lithographic skull reconstruction makes it possible. In types I and III not only is the condyle enlarged but its neck is also. It is thicker and longer.

In type II H.E., no real condylar hyperplasia is present. The condyle is not noticeably enlarged. It can be within the normal range in size and shape. In its slender form it might give the impression that the condyle is on the smallish side, but this is probably only due to the elongated narrow neck. In these cases the condyle often presents in a form as if it were only the upper end of the neck.

11.5**Hypothesis of Pathophysiology**

As mentioned in the chapter “The Condyle and its Functions” I believe that these asymmetries develop due to

two different growth regulators which lie within the cartilaginous section of the condyle. The one is responsible for growth in length (growth regulator or factor L) of that hemimandible and the other for growth in mass of bone (growth regulator or factor M). Only by postulating the existence of two such growth regulators can we elucidate the development of these form anomalies. And only through their existence can we explain why the abnormal growth of these anomalies should immediately cease when a high condylectomy is performed on the affected side.

It also seems that these two regulators produce different histological pictures as shown by H. Obwegeser and M. Makek (1986a,b) in their original publication.

If the assumption of the existence of two different growth regulators or growth factors is correct, then it is not difficult to imagine that they can be in normal balanced physiological activity and as such allow the genetic pattern of each mandibular half to develop. But their existence also makes it understandable that only one of them may become hyperactive, for whatever reason, and then produce a surplus of that growth for which it is responsible, either as surplus in length or in mass.

Consequently, when only one growth regulator is hyperactive in one condyle, the pure form of that asymmetry will develop. It will also be understandable that the hyperactivity can be very mild, thus producing a rudimentary asymmetry of that corresponding form. On the other hand the hyperactivity may be very "wild". Then an excessive growth of the corresponding type will occur very rapidly.

No doubt, in any one condyle, not only one of the growth regulators can be hyperactive. Both growth regulators can be hyperactive at the same time and yet be different in intensity.

When both regulators in one condyle are hyperactive, a hybrid form will develop with surplus of growth in length and volume in the same mandibular half as compared with the contralateral normal half. Also, in these hybrid forms, the two regulators may not be equally hyperactive. This means that in hybrid forms of H.H. and H.E., almost always one or the other regulator predominates. Therefore, there is a chance of a hybrid form with a 50% contribution of each regulator or a very unequal distribution such as 80%:20%, or any other combination. This is very interesting. One might expect that the stimulation to hyperactivity would be the same for both growth regulators, whatever the cause of the stimulation might be. This is not the case. The one or other can be more active. The picture of the anomaly will accordingly be predominantly either H.H. or H.E.

This fact suggests that the stimulus for hyperactivity of the growth regulators L and M cannot be the same. The stimulation seems to lie within the growth regulator itself. If not, it will not be understandable why the

one reacts differently to the same stimulus as does the other.

As these growth regulators in the condylar cartilage must exist, naturally it can also happen that on both sides of the mandible such misregulation of growth can develop either the same type or the other one, or even a mixed form. When they appear bilaterally they are practically always asymmetrical. This is true also in those cases of hyperactivity in which the same regulator type is responsible for the anomaly. In such cases it is the intensity of hyperactivity of the regulator responsible which is not equal on the two sides. This fact, and in particular the unilateral form, proves that the abnormal growth mechanism does not follow central direction. Also, the observation of an immediate cessation in abnormal growth after high condylectomy is additional evidence. The cause for such a hyperactivity of the growth regulator within the cartilage zone must be a local one. We are of the opinion that each activation, if it were mediated via a central growth steering mechanism, would always affect both sides equally. The classic example and proof is acromegaly. In acromegaly the mandible is the only bone in the whole skeleton which can become longer. From this fact, one might deduce the consequence that the pituitary hypophysis must stimulate these condylar growth regulators into hyperactivity. But there is no proof for that. Not in all cases of acromegaly does the mandible become longer (A. Künzler 1988). However, it seems possible that the hypophysis can activate mainly either growth regulator M or L. In one of our acromegaly cases I found a H.E.-like abnormality on one side and a H.H.-like shape of the mandible on the other side. Unfortunately, the documentation of this case is missing.

The three different forms of these mandibular asymmetries and their various degrees of expression will be demonstrated separately. Differential diagnostic problems as well as therapeutic guidance will also be discussed.

11.6 Aetiology of Condylar Hyperactivity

Trauma has often been mentioned as a possible aetiological cause (W. Lineaweaver et al. 1989; P. Hyckel et al. 1991; J. Skolnik et al. 1994; among others). Sometimes the patient or parents will mention a fall on the chin in early childhood as a possible precipitating factor in the growth of the anomaly. If that were a possible cause of condylar hyperactivity then it would have to start soon after that incident. I have neither read nor heard of this in relation to any of the three forms. Of course, it cannot be denied that prepubertal trauma can lead to mandibular asymmetry. But I have seen it only as an aetiology for condylar hypoactivity, with condylar and hemimandibular hypoplasia as a consequence.

Inflammation could be considered as a cause for such a hyperactivity. But on the contrary, it always results in hypoactivity.

Hypervascularization has also been considered as a possible cause of condylar hyperplasia (T. Øberg et al. 1962; R. Walker 1967; P. Egyedi 1969). But the question arises whether the hypervascularization is the cause of the condylar hyperactivity or whether it is the result of it. As there is cellular hyperactivity present, provable by the use of radionuclides, I think the egg was first and not the chicken.

Genetic influence might also be considered as a possible aetiology. I have not seen a single case of H.H. in which inheritance was provable. But there are reports of proven genetically inherited aetiology for mandibular asymmetry of the H.E. type (N. Rowe 1960; R. Walker 1967; M. Person 1973). Unfortunately, in their publications, these authors do not provide sufficiently good documentation. Apart from the patient's outward appearance, demonstration of their occlusion and radiographs would also be necessary to make a clear diagnosis of the type of the anomaly. It is therefore not possible to say whether they refer to an unilateral or bilateral H.E. type or an unilateral hypoplasia type.

I myself have not seen a single case of pure unilateral H.E. of clear hereditary origin. But asymmetric prognathism often has the physical features of a bilateral H.E. and sometimes its hereditary origin does not seem to be deniable.

Whether it will ever be possible to verify the existence of these two growth regulators by experimental work I do not know. I am afraid I will not live long enough to enjoy the pleasure of such a verification of my hypothesis on the existence of the two condylar growth regulators which can produce three different types of mandibular growth anomalies.

11.7

General Surgical Treatment Considerations

When discussing the philosophy of treatment planning for the various forms of mandibular growth anomalies, we have to distinguish between the different forms. Condylar hypoactivity, of whatever origin, will require an entirely different type of correction to the condylar hyperactivity forms.

The *condylar hypoactivity cases* need reconstruction to compensate for lack of growth; this is often done by unilateral or bilateral advancement of the mandible by the use of a sagittal splitting of the rami procedure. Some cases may require a costo-chondral graft to be inserted, early in life, to avoid the lack of downward growth of the maxilla on the hypoplastic side. Cases with gross deficiency of the condylar-ramus area, of congenital origin, or after osteomyelitis may need more extensive reconstructive work.

It is not necessary to discuss here all the various problems involved in such cases. The various cases will show my way of dealing with them.

For me the question of whether or not it would be better to remove the upper half of the condyle very early in a hypoactivity case as we do in the hyperactivity ones and let the "functional matrix" be responsible for further growth has not yet been answered. We do know that in all unilateral condylar hypoplasia cases the growth results in a hemimandibular hypoplasia. We also know that if a damaged condyle is present, it will not guarantee normal growth when function is reduced but that a normal undamaged condyle does allow that mandibular half to develop normally in spite of complete ankylosis of the contralateral side. And we also know that the affected side of the mandible will develop normally when we remove a traumatically damaged condyle in early childhood and restore normal function. This seems to indicate it would be logical to perform an early high condylectomy in those cases of condylar hypoactivity which are not due to trauma, but have normal function. I suggest that this should be tried. As I myself am no longer in clinical practice, I cannot do it myself, regrettably.

In the *condylar hyperactivity cases* we have to distinguish between H.H. and H.E. and the hybrid types and we also have to distinguish between a still active case and the case in which the hyperactivity has burned itself out. In all three types we can "wait and see" when there is rudimentary hyperactivity only. But even in a mild case one should not perform corrective osteotomies before being absolutely certain that the hyperactivity is burned out, independent of the patient's age. Otherwise one runs the risk of relapse (G. Hampf et al. 1985). However, for psychological reasons, it might become necessary to correct a severe asymmetry earlier. Then the patient must be informed that a second operation may be necessary at a later date.

In the *H.H. and in hybrid types* it might be necessary to perform an early high condylectomy as soon as the diagnosis is made and when there is clear evidence of a tendency to progress. As we never know the final extent of disfigurement, it is better that we arrest it whenever we are in doubt.

It is not always that easy to produce a perfect result in a severe H.H. or hybrid type case. The more rapidly the asymmetry and with it the height of the horizontal ramus and the asymmetrical facial disfigurement develops, the more complex becomes the surgery that will be necessary. The main object is to arrest further abnormal growth on the affected side (J. Howell 1963; R. Walker 1967; J. Delaire et al. 1983; G. Hampf et al. 1985; A. Gordeff et al. 1988).

In the *H.E.-types* of growth anomalies one not only has to distinguish between the active and non-active case, but also between the unilateral and bilateral ones.

In a very highly active unilateral case, early high intra-capsular condylectomy is indicated. The slowly developing H.E. case can wait until growth has ceased.

The bilateral cases of H.E. present a more complex problem, as in these cases often the maxilla has also to be included in the treatment planning. Again a very fast growing bilateral H.E. case may require an early uni- or bilateral high condylectomy. In a more moderately developing case I rather tend to wait until growth of the maxilla as well as the mandible has come to a stop. After that the treatment planning is the same as in any other non-hyperactivity cases.

Chin correction becomes necessary in a large percentage of asymmetric mandibular growth anomalies, in cases of condylar hypoactivity as well as in cases of hyperactivity. The correction may be necessary because of an asymmetry and/or any other type of chin anomaly.

Contour asymmetry correction is often necessary in addition to corrective osteotomies. It might be in the ascending ramus-body region, in the chin region and also in the paranasal and zygoma areas. For this purpose I like to add slices of lyophilized human cartilage subperiosteally or a carved piece. The thin slices, cut with a dermatome, have an advantage over the insertion of a block of cartilage. The block itself may distort after some time and then alter the desired result. The thin slices may be placed as single pieces one beside the other or they may be fixed together with a resorbable suture. Because they are so thin they will be fixed to the bone by fibrous tissues which develop between all the implanted slices (H. Sailer 1979). If it is found that there has been an over-correction, the surplus can easily be removed under local anaesthesia.

For the *correction of muscle or bone surplus* the transoral approach is also the only acceptable one. In the chapter "Main Standard Operation Techniques and Instruments" details are described under "Transoral correction of masseter hypertrophy".

11.8

General Orthodontic Treatment Considerations

Usually all these cases of abnormal mandibular growth regulation are first seen by an orthodontist. This is at least true for those cases which start during the growing period (this is the largest group). Therefore, the orthodontist takes the main responsibility and burden for the patient's final good result. This means that the orthodontist has to be familiar with the pathophysiology of all the cases of condylar hypo- and hyperactivity.

In the hyperactivity cases, the orthodontist often tries to arrest the asymmetrical growth by producing as good an occlusion as possible. In my experience these abnormal growth activities cannot be stopped by ortho-

dontic means. It is a waste of time and money if one tries to deal with clear condylar hyperactivity in a conservative way.

It is my opinion that for every asymmetrical facial or mandibular development the cause must be determined. Only through this knowledge can the correct treatment be planned. For necessary clarification, not only the patient's history, physical findings and proper radiographs be investigated, but also scintigraphic investigation may be necessary. Often, of course, it is wise to see the patient again in six months and compare his outward appearance, his cephalometric and other radiographs and his occlusion with the previous examinations. But I have also learned, several times, it would have been better to have seen him again in 4 weeks and then immediately performed a high condylectomy. In most cases it is worthwhile for the orthodontist to contact his surgical colleague as early as possible.

Orthodontic treatment for all three forms of condylar hyperactivity should consist in achieving nothing but correct positioning of the teeth onto the respective base of the jaw of both the mandible and maxilla, independent of necessary extractions, in such a way that the surgeon can achieve a good occlusion after mobilization of the jaws. The same is true for the hypoactivity cases.

In the unilateral cases of H.E. it is not rare for the teeth to erupt and become arranged spontaneously in such a way as if the jaws were in proper relationship and the position of the teeth and intercuspitation were guided by the occlusion. This is in spite of the fact that the mandible is shifting progressively over to the normal side, producing a more or less severe crossbite. In these cases the models of the teeth can be brought into proper intercuspitation without any pre- or postsurgical orthodontic treatment. Any orthodontic attempt to stop the unilateral overgrowth will result in unfavourable arrangement of the teeth.

Often enough, I have experienced the situation where an orthodontist had treated the young patient for several years and had tried hard to bring the teeth into an acceptable occlusion. Finally, he referred the case when he saw that he could not overcome the force of asymmetrical growth. Most of these cases turned up with a teethposition situation which did not permit me to move the jaws to the place where they were needed for good intermaxillary and profile relationships. The orthodontist and the patient had to start again to position the teeth in such a way that surgical correction of the asymmetry would become possible.

The patient must be informed that in spite of presurgical orthodontics final adjustments may have to be made postoperatively.

In cases of H.E., whether unilateral or bilateral forms, it is best to do no orthodontic treatment at all until surgery is planned, except taking care to prevent tooth crowding by necessary extractions. The surgeon has to

produce a symmetrical mandible. That might involve more than just bringing the teeth together.

I feel strongly that it is wrong, for an orthodontist to do his presurgical arrangement of the arches of the teeth without contacting the surgeon beforehand, and then tell the surgeon what he should do. Often enough I just could not accept the orthodontist's plan for surgery. When the orthodontist prepares the case alone according to his philosophy, the surgeon may be forced to do what he, the orthodontist, wants. But the final and lasting results are the responsibility of the surgeon, for now and for ever! The worst scenario is overexpansion of the maxillary arch and retrusion of the lower front teeth.

11.9

Prognosis

In my experience it is not possible to inform the patient how long the abnormal growth will continue, except that it will be as long as the condylar hyperactivity or the increasing hypoactivity continues. Although in most cases it will end around the end of normal growth, this cannot be promised. So, a prognosis cannot be made without the risk of giving misleading information to the patient. Rarely, but at any time, a mild degree of hyperactivity can all of a sudden become more severe.

Hemimandibular Hyperplasia (H.H.)

12.1

Historical Cases

The largest number of cases ($n=66$) has been studied by M. Rushton (1951). He included in his paper interesting historical cases from the collection of the Royal College of Surgeons, not only of H.H. but also of hemifacial hyperplasia and of acromegaly.

J. de Burgh Norman and D. Painter (1980) published a very interesting historical review on the condylar hyperplasia anomaly. However, according to the illustrations presented and the patients histories of these ancient cases it is quite obvious that some of them were not H.H. anomalies due to true condylar growth hyperactivity. Rather they had developed a tumour-like irregular enlargement of the condyle due to a generalized arthritis or other causes. As in those days no radiographic pictures of the affected mandible were available, our diagnosis can be made only on the drawings of the shape of the patient's appearance and their mandibles, as the surgeon of those days imagined the mandible would look like. A gross enlargement of the condyle, of whatever the origin, will produce almost the same facial appearance. But true H.H. or the hybrid form of H.H. and H.E. will always have an increase in height of the mandibular body, not of the ascending ramus only, and a downward convexity of its inferior border on the affected side. Both of these signs do not exist in cases of enlargement of the condyle from any other origin. By radiography or by palpation these symptoms could be recognized. This means that we cannot say that the facial asymmetries of these ancient cases are caused either by a true H.H., or a hybrid form, or a tumour-like enlargement of the condyle, or of any other origin.

12.2

Unilateral Hemimandibular Hyperplasia

12.2.1

Clinical Appearance of Unilateral H.H.

Unilateral H.H. is characterized by a three-dimensional enlargement of one side of the mandible, i.e. the enlargement of the condyle, the condylar neck and the ascending and horizontal rami. The anomaly terminates

exactly at the symphysis of the mandible. That is why we call this anomaly, which affects one half of the mandible, "hemimandibular hyperplasia".

Clinically, the following details are discernible: in pronounced cases the striking increase in height on the affected side and the medial shift of its mandible and the lateral and upward push of the contralateral mandible gives the face a rotated appearance. The unilateral asymmetric increase in height of the face, even when not well-marked, gives rise to a sloping rima oris. The mouth can be opened without restriction and the sloping rima oris and the facial asymmetry still remain discernible, even if they are less marked. The corner of the lip commissure on the affected side is displaced inferiorly only and not laterally also. This is in clear contradistinction to hemimandibular hypoplasia, in which the corner of the lip commissure is more lateral and higher than on the normal side.

Viewing the face from below discloses the anatomical cause of the facial asymmetry: a very striking unilateral downward and medial projection of the ascending ramus and the angle and in particular the whole horizontal ramus. Looking at the patient from in front or from below, a marked flatness of the cheek area on the affected side becomes very obvious. In spite of the height increase on the affected side, the mandibular body seems shifted towards the facial midline, thus creating a difference in width of the two facial halves. The affected side is closer to the facial midline than is the normal side. Since the anomaly normally commences before puberty, it is understandable that the maxilla, including the teeth and the maxillary sinus, follows the downward growth of the mandible. This results in a tilted occlusal plane. The teeth on the non-affected side of the mandible always tilt towards the abnormal side and yet seem to try to remain in occlusion with the upper teeth at a higher level than those on the abnormal side. Because of this tilting, the crowns of the teeth on the normal side appear shorter than those on the abnormal side. Those on the abnormal side seem to be in a more vertical position than normal and can occlude nicely.

The contralateral side of the mandible will also be influenced by the downward growth on the affected side, although the abnormal growth clearly stops at the symphysis. The lingual tilting of the lower alveolus with its

teeth on the normal side, is caused by the lateral and upward rotation of the normal mandibular half, due to the downward growth of the affected side.

The upper dental arch on the unaffected side can grow downwards by-passing the lower teeth when they are very severely lingually inclined, ending up in buccal non-occlusion causing impressions on the lower gingiva. This gives rise to a rotated lower dental arch which is a reflection of the similarly distorted facial appearance of the H.H. case.

There is another finding noticeable on the normal side: the downward and medial growth of the abnormal side of the mandible seems to produce so much "pressure on the mandibular body of the other side that it not only rotates its lower border laterally and upwards, but also reduces the height of that mandibular half, including the alveolar process, so much that it becomes obviously less high than a normal mandible. The more the abnormal side increases in height, the more the body of the mandibular half of the normal side will be rotated outwards and upwards while the alveolar process with its teeth rotates lingually and yet tries to remain in contact with the teeth of the maxilla. The result is a typical corkscrew-like, twisted appearance of the dental arches. The pressure from the affected side on the normal side causes a rotation of the mandible on the non-affected side.

Therefore the increase in height on the abnormal side leads to a decrease in facial height on the normal side. This bilateral facial height difference results in a typical H.H. appearance.

In the H.H. cases, the oblique occlusal plane has a direction contrary to that in a hemimandibular hypoplasia case. In the H.H. case it is displaced inferiorly on the affected side and consequently requires upwards repositioning. In a hypoplasia case, the maxilla cannot grow downwards enough because the height of the ascending ramus is too short. It will need surgical inferior repositioning on the affected side. The degree of obliquity of the occlusal plane depends, in both cases, on the developmental stage of the maxilla when the hyper- or hypoactivity commences and also the intensity.

The elongation of the ascending ramus and the caudal displacement of the horizontal ramus, which take place entirely unilaterally with a clear demarcation at the symphysis, can give rise to a kink in the mandible at its symphysis. This is also clearly demonstrated by viewing the mandible from below and, in particular, on the panoramic radiograph (see Fig. 31h).

The hyperactivity in the case of H.H. can lead to such a rapid downward growth of the affected side that the maxilla cannot follow as rapidly and an open bite will develop on that side.

Such cases give the impression that first of all the hyperplasia type produces a downward growth of the ramus and that consequently the downward convexity of

the mandibular inferior border and the large distance between it and the teeth are compensatory consequences. This could indicate that the teeth grow upwards in order to occlude with the maxillary teeth which inferiorly cannot follow rapidly enough. However, the fact that the mandibular canal, in very severe cases, is always closer to the inferiorly bowed mandibular lower border than found in normal cases (it seems almost pushed downwards), leads to the conclusion that the H.H. deformity also includes an excessive production of cancellous bone. Also, the downward convexity of the inferior border of the mandible seems to be a product of the anomaly itself and not just a secondary deformity because of the vertical elongation of the ramus. If that were not so, we would also find inferior border convexity and additional excessive cancellous bone production in other aetiological cases of vertical height increase in the ascending ramus area. Neither in tumorous nor tumour-like causes of ramus height increase nor in cases of H.E. have we observed this. Therefore, the inferior border convexity as well as the increase in alveolar height must be a direct product of growth regulator M hyperactivity. While we have an increase in mass in the condyle and neck and ramus with definite vertical elongation of these structures and increase in height of the horizontal ramus, there is no elongation of the horizontal ramus, except in hybrid form cases in which the L-regulator is also hyperactive, not the M-regulator only.

12.2.2

Radiographic Findings of Unilateral H.H.

Radiographically, the panoramic view with the teeth in occlusion seems to be the best projection. As seen in Fig. 31h and Fig. 37i, it reveals typical pathognomonic findings: the ascending ramus is clearly elongated in all its sections. This means that the distance between the depth of the semilunar notch (IS-point) and the angle (AG-point) (called the trunk of the ramus), is clearly larger than on the normal side. In this classical case of a medium degree of H.H., the difference in trunk height between the normal and abnormal side (10 mm) is almost 20%. In addition, the neck of the condyle is longer by 3 mm (almost 30%). The height of the condyle differs by 4 mm. Altogether the left ramus including condyle and neck measures 87 mm while the normal side 70 mm. That is a difference of 24% between the normal and hyperplastic ramus including the condyle. The height of the horizontal ramus on the hyperplastic side measures 34 mm between the lower border and the crest of the alveolus in the interdental region between the first and second molars. The height of the normal side in the same area is 25 mm. That is a difference of almost 40%. The real height of the base of the mandible is very difficult to determine. The inferior border of the base is clear but not so the upper line of the mandibular

canal in the vertically enlarged horizontal ramus and even less in the same area on the normal side. The width (breadth) of both ascending rami is almost equal and the thickness is not measurable in this projection. In the p.a. projection of the mandible the difference in thickness is also very clearly recognizable, measuring 3 mm thicker on the abnormal side in this case. But this measurement might be influenced by the X-ray technique. Computerized tomograms would be more accurate.

The angle is characteristically rounded off and the mandibular lower border is bowed downwards and positioned at a lower level than that on the other side. In the angle area and in the posterior half of the body, the cortical plate is often very thin or even not clearly recognizable, contrary to normal. The downward curvature is best seen in the bicuspid region. The essential increase in height of the horizontal ramus is expressed by the increased distance between the tooth root apices and the inferior mandibular border. The mandibular canal is displaced down towards the lower border of the mandible. In this region it seems that only the alveolar process itself is increased. The quality of its trabeculation differs from that of the normal side. The trabeculae are coarse, thicker and wider apart when compared with those on the normal side. The horizontal ramus of the contralateral side always shows a relatively reduced vertical height, in the area of the molars and premolars. The enlargement of the affected side of the mandible which terminates exactly at the symphysis can be clearly seen, especially in the orthopantomogram (see Fig. 37i). It can also not be missed on the postero-anterior view of the mandible. The elongated ascending ramus and the coarse, thickened and wide-meshed trabecular structure of the affected side are often particularly obvious in these views (see Fig. 37l, m). This difference cannot be missed in all X-ray projections when compared with the normal side. The elongation of the ascending ramus and the increased height of the horizontal ramus, together with the compensatory downward growth of the maxilla with its sinus, constitute the essential skeletal basis of the vertical increase in height of the affected side of the face producing the facial asymmetry. In pronounced cases, these skeletal changes which take place essentially unilaterally, bringing about the well-known and typical rotation effect on the face previously mentioned.

The very enlarged condyle is usually irregularly deformed; the condylar neck is thickened and elongated and occasionally kinked. All these findings are particularly noticeable in comparison with the unaffected side. The wide-meshed coarse trabecular structure is found in the condyle, the neck, the ascending ramus and the horizontal ramus. Some trabeculae appear as if they were sclerotically enlarged, a picture which is always seen whenever a noxious stimulus, be it inflammation, a malignant osteoblastic tumour, irradiation or any other cause, gives rise to osseous restructuring and production. The structure is very similar to the radiographic findings in uninfected areas of irradiation damage to the mandible. The ascending ramus and condyle of the unaffected side appear especially fine structured. This might be partially due to the thrust exerted by the enlarged side.

There is still something to be mentioned regarding the position of the mandibular canal in relation to the mandibular lower border: the faster the asymmetry develops the closer to the mandibular lower border is the mandibular canal found (see Fig. 31h). In cases of rather or very slow abnormal unilateral growth, in particular when it goes on for several years, even into adulthood, the mandibular canal is found further away from the lower border and it is easily visible (see Fig. 32h).

12.2.3

Variations in Unilateral H.H.

The hyperactivity of the condylar M-growth regulator is not of the same intensity in all cases, nor does it always start at the same age and go on for the same length of time. Although, unilateral H.H. will always appear with the same classical symptomatology, clinically as well as radiologically, its appearance can vary quite a bit, depending on the patient's age when it starts, its intensity and duration. Only when the investigating orthodontist or surgeon keeps these facts in mind will he be able to understand the different pictures and details of the radiology. It is for this reason why a number of varying cases are presented. None of these cases is equal to another. I have tried to avoid presenting the same situation in more than one patient.

Unilateral, Pure H.H. of 12 Years' Duration (Fig. 30)

Sex and Age. Male, 38 years of age.

Aetiology and History. Beginning in childhood about the age of 10 years, the left side of the face gradually became longer. It developed rather slowly and resulted in the present appearance at the age of 22. The patient does not remember any possible cause for its origin. With the increase in severity of the facial asymmetry, the occlusion became more and more distorted. The right upper teeth occluded laterally to the lower ones, impinging on the gingiva. Eating became more and more difficult. The patient sought improvement in his occlusion as well as his facial appearance.

Clinical Findings. Very pronounced vertical facial height asymmetry between left and right sides (Figs. 30a–c). Both halves end exactly in the midline of the chin prominence which is precisely in the facial midline. The right side seems less in height than normal while the left side is much longer than usual. The lip commissure is oblique, tilting down from right to left. The left lip corner is much lower than the right. Musculature and facial innervation are normal. Mouth opening is not reduced, but it ends with a deviation to the right side.

The situation of the teeth cannot be described as an occlusion (Fig. 30d). Some upper front teeth on the left side are missing. The right upper incisors and the canine pass the lower ones laterally and reach their gingiva. The left lateral teeth are somehow in occlusion but the upper

as well as the lower ones are further inferiorly than they should be, creating an oblique occlusal plane.

Radiographs. At the time when this patient was seeking improvement of his problems, there were no panoramic projections available. *Ordinary lateral views, TMJ tomograms* and in particular the *p.a. projection* (Figs. 30e–g) of the mandible disclose the mandibular abnormality very clearly. The increase in vertical height on the left side is particularly clearly visible in the p.a. projection of the mandible. This is due to an increase in ramus height including the condyle and its neck and is also due to an increase in the height of its mandibular body. The condyle is clearly enlarged but moves forwards normally onto the articular eminence as does the normal condyle on the right side. In spite of its enlargement its shape was not deformed. It is difficult to evaluate the trabecular structure on these radiographs. The mandibular canal seems to run at a fairly good distance from the lower border of the mandible which has a strong cortical plate.

Provisional Diagnosis. Unilateral pure H.H.

Treatment Plan. Resection of the whole left condyle – only partially for height reduction of the left side – mainly for histological purposes, and a sagittal splitting procedure of the ramus for lengthening on the right side in order to correct the vertical shortness as well as

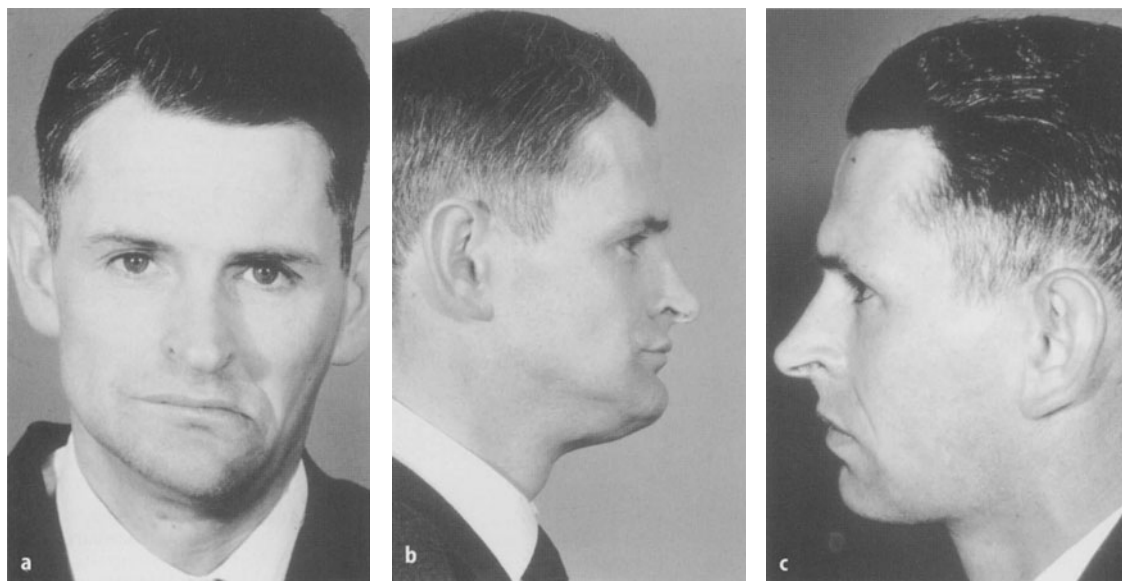


Fig. 30a–c. Unilateral pure H.H. **a–c** Front and profile views are typical. No deviation of the chin prominence

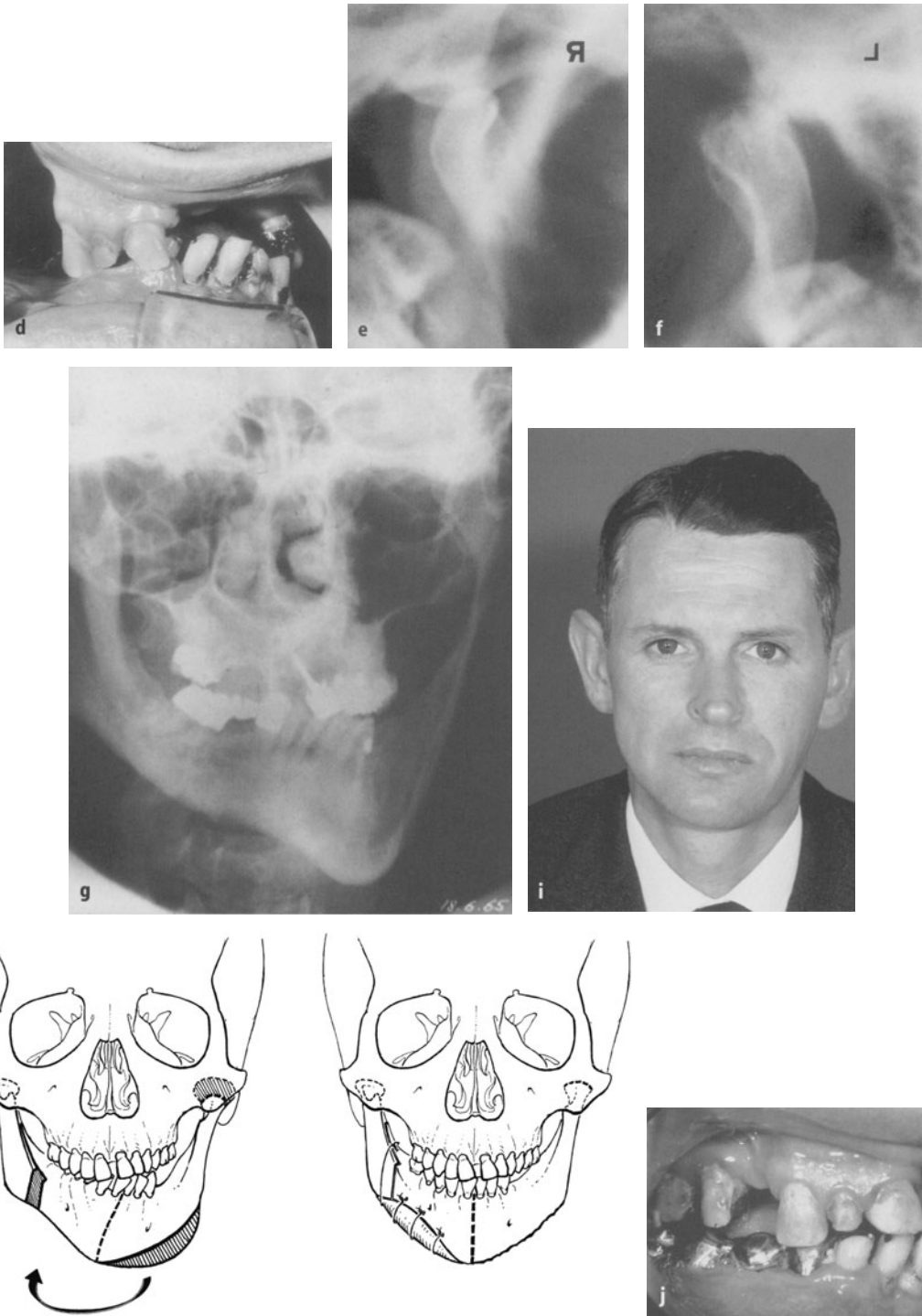


Fig. 30d-j. Unilateral pure H.H. **d** In spite of the lack of several teeth, it is typical of very severe cases that the upper teeth may pass the lower on the contralateral side and touch the gingiva, partially due to the medial rotation of the horizontal ramus and partially due to downward growth of the maxillary teeth. **e-g** The tomograms of the TMJs disclose the typical difference in size and shape of the two condyles and so does, even more impressively, the radiograph with the mandible in p.a.-projection. **h** Diagrammatic illustration of the planned surgical correction. **i, j** The surgery resulted in a symmetrical face and a relationship of jaws, such that prosthetic reconstruction had become possible

to permit correction of the lateral overbite on the right side. The left mandibular nerve should be freed and the convex lower border on the left side must be resected and fixed to the right lower border to increase the vertical height deficiency of the right mandibular body (Fig. 30h).

Actual Treatment and Result. The surgery was carried out as planned, with 6 weeks of intermaxillary fixation postoperatively. The resected condyle had a rather irregular surface, as often observed, in benign tumours.

An absolutely normal appearance was achieved and the occlusal situation was improved so that prosthetic

reconstruction of the occlusion and teeth had become possible (Figs. 30i,j).

Discussion and Conclusion. The classical form of pure unilateral H.H. can easily be diagnosed without present day sophisticated radiological examinations. However, without them we miss a lot of desirable information in cases which are not all that clear. The length of time of abnormal growth seems to permit the mandibular nerve to stay away from the convex lower border which makes corrective surgery easier. The main goal for the patient is the normalization of his facial asymmetry and of his very severely disturbed occlusion.

Unilateral, Pure H.H. of Medium Degree, Active for 4 Years (Fig. 31)

Sex and Age. Female, 16 years and 6 months.

Aetiology and History. Four years previously, at the age of about 11 years, the patient's parents had noticed the development of slight asymmetry of their daughter's face. No aetiological factor could be found, there was no recollection of trauma and the family history was also negative. The asymmetry had gradually increased. An orthodontist was consulted who felt that there was no indication for presurgical orthodontic treatment because of the existing class I occlusion. As the girl was worried about her facial asymmetry she was referred for corrective surgery. She did not complain of any pain.

Clinical Findings. The facial appearance of this young female patient shows the very typical deformity of a medium degree H.H. (Fig. 31a–c). The face is asymmetrical, with the left side remarkably longer than the right side. The lip commissure is slightly oblique with the left corner lower than the right. The prominence of the chin is exactly in the midline of the face. The facial midline is the turning point of the inferior mandibular contour. This passes downwards and medially on the long left side and upwards and laterally on the slightly too short right side. The difference between left and right sides is clearly seen in the front view of the patient's face as well as from below. The difference in the facial height between left and right is also very obvious in the two profile views. Mouth opening is not impaired and there is almost no noticeable deviation to either side (Fig. 31d). Musculature, sensory and motor nerve function in the face were unremarkable.

The occlusion shows the typical picture of a unilateral H.H. of medium degree (Figs. 31e–g): In the front view, the lower interdental midline is tilted towards the hyperplastic side with the full dentition in central occlusion. It is a typical sign of unilateral H.H.: well-erupted teeth on the abnormal side and apparently shorter

teeth on the "normal side", due to their slight lingual tilting. The lateral occlusal view also demonstrates this difference very clearly.

Radiographs. *Panoramic view* in closed occlusion (Fig. 31h): this X-ray projection for diagnosis of the shape of the mandible shows the difference between left and right sides very clearly. There is a remarkable increase in volume of the left hemimandible. This increase includes the size and shape of the condyle and its neck. On the radiograph the left ascending ramus seems essentially equal to the right one but in measuring there is a clear difference between left and right. The left angle of the mandible is well rounded with a much larger radius than that on the right side and also than that of a normal one. No cortical plate is visible around the angle and ascending ramus. The lower border of the left mandible is much lower than that on the right side. There is a difference in height between left and right bodies of 9 mm in the region of the first and second molars. The lower border on the left side has a clear convexity. The right lower border is in an almost straight line between the angle and the midline of the chin where the difference between left and right sides is marked by a slight concavity. There is also a difference in structure between left and right mandibles: the left hyperplastic side shows a rather coarse trabeculation in all sections, body ascending ramus, condyle and neck when compared with the normal right side. The canal of the mandibular nerve (as the nerve was never in the alveolus but in the body of the mandible I prefer to use the term mandibular nerve, as already mentioned in the chapter "The Mandible") passes, on the hyperplastic left side, very close to the lower border, just adjacent to the very thin cortical covering, lower than is normal. In this radiograph, the mandibular canal on the right side can

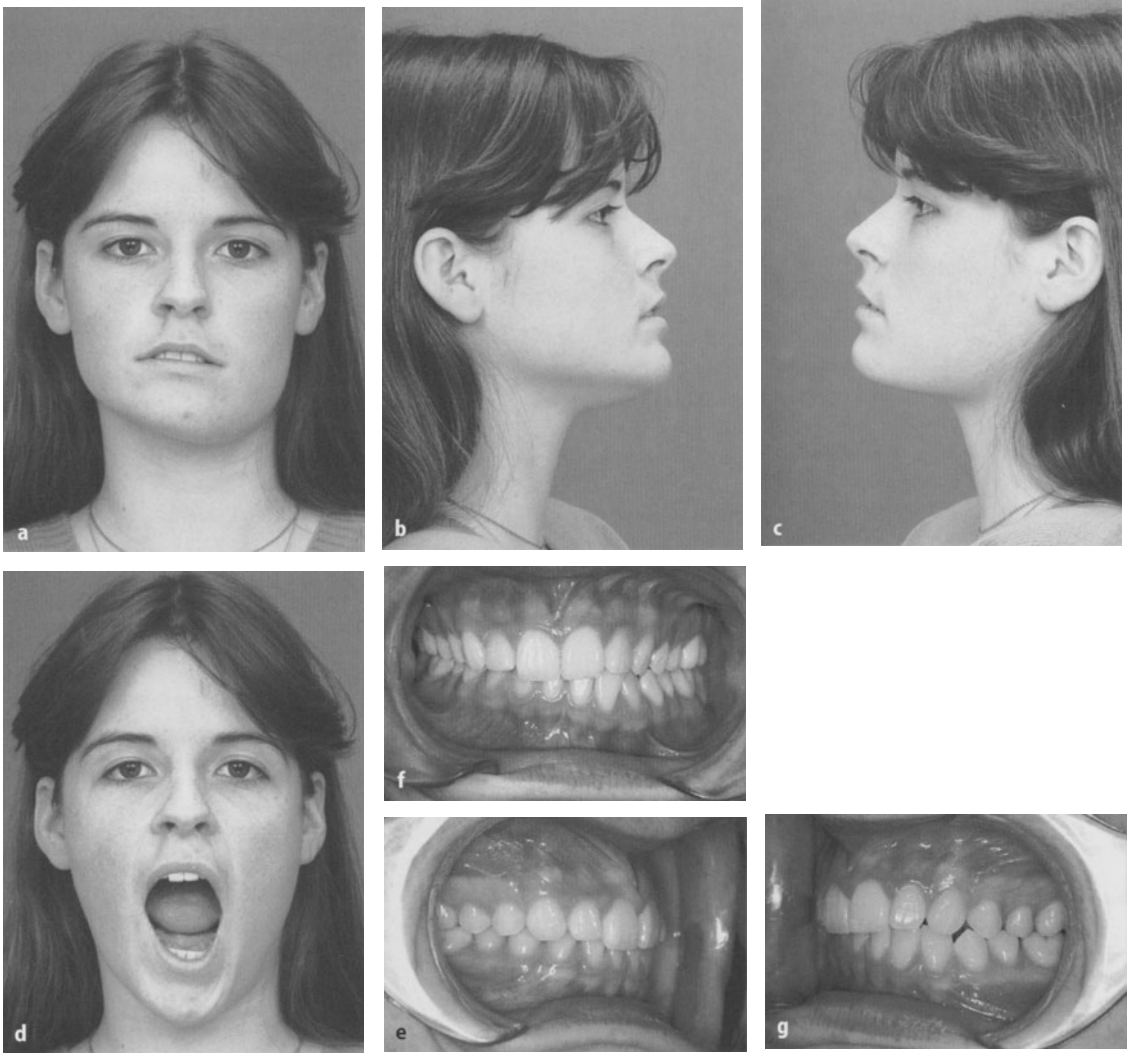


Fig. 31a–g. Unilateral classical pure H.H. of medium degree, active for 4 years. **a–d** Typical facial appearance of unilateral H.H. of medium degree. **e–g** Typical occlusal picture of unilateral H.H. of medium degree

only be visualized as far as the not yet fully developed third molar. The mandibular teeth are all present. The axes of all of them are directed slightly towards the mid-line. However, the lower incisors are clearly tilted slightly towards the abnormal left side. If one draws a line between the highest points of the two articular fossae then a very remarkable difference in height between right and left halves of the maxilla can be recognized. This difference is true also for the inferior extension of the maxillary sinus as well as for the occlusal surfaces of the teeth. As the upper and lower teeth are able to occlude, a bit of an oblique occlusal plane results, coinciding with the oblique lip commissure. The articulation is a normal class I-type.

The TMJ tomograms including the ramus (Figs. 31i, j) also show clearly the difference in shape, size and trabeculation between left and right sides. In particular, the condyle and its neck are much larger, but only slightly longer on the left side than on the “normal” opposite side. The condyle itself is almost round. Also the difference in the trabeculation type can be recognized.

The lateral cephalogram (Fig. 31k) as well as the *mandible p.a.* in closed position (Fig. 31l) also demonstrate clearly the above mentioned differences in height, shape and structure of the two mandibles as well as the slightly oblique occlusal plane. *The p.a. projection of the mandible* in the maximum opening position (Fig. 31m) demonstrates clearly the vertical elongation and in-

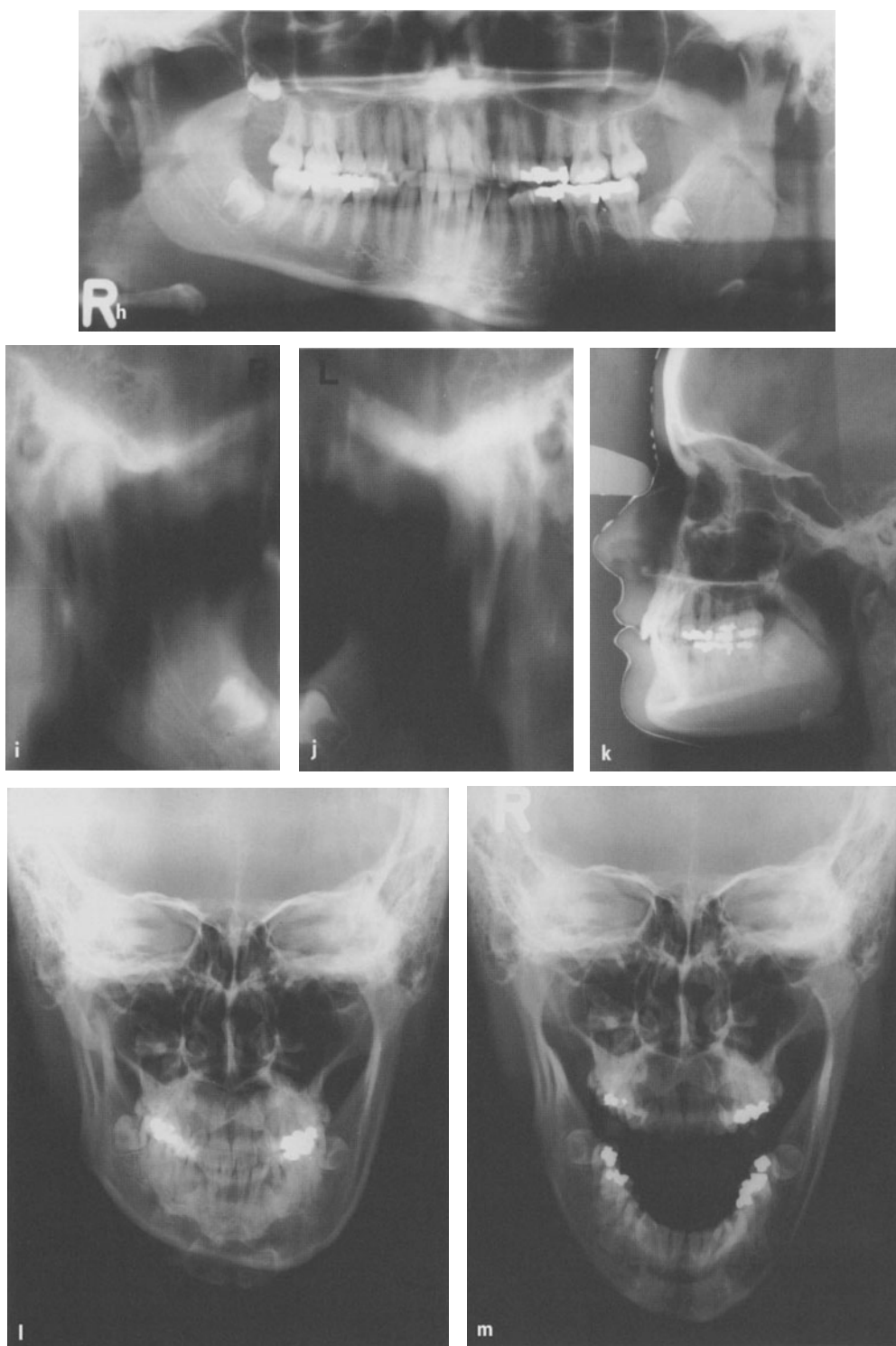
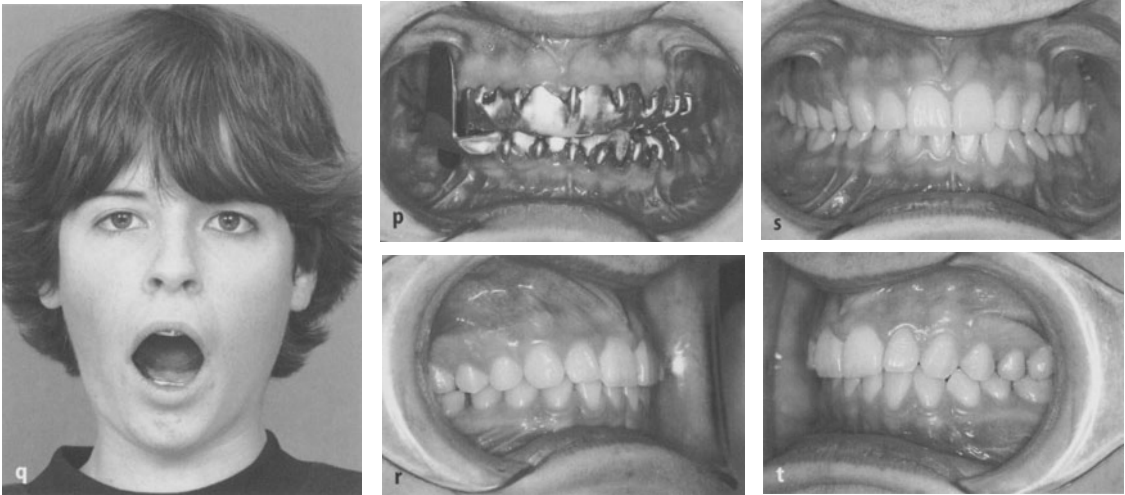
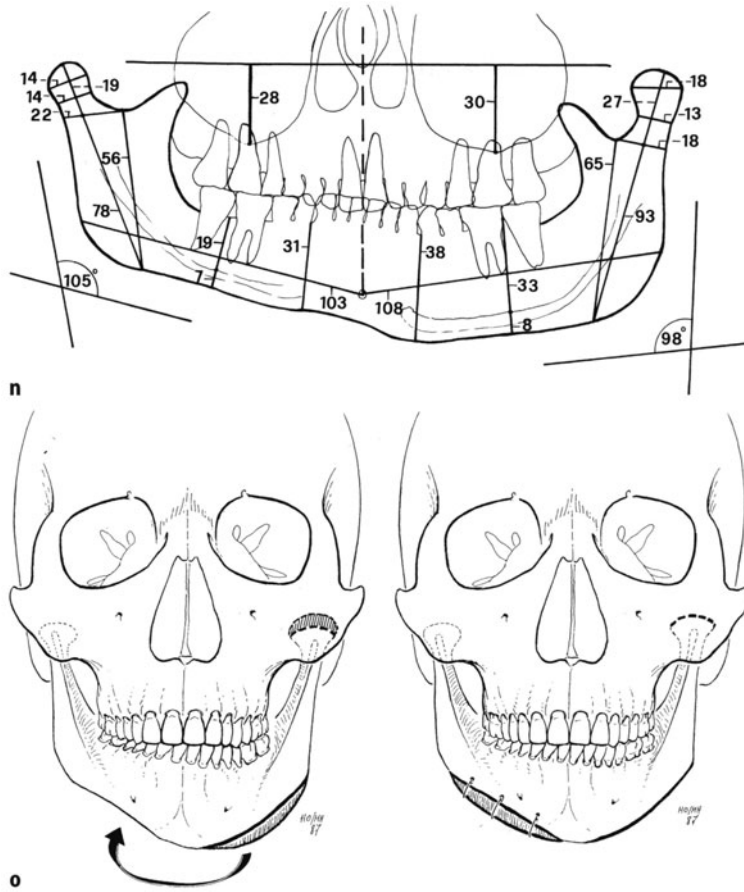


Fig. 31h–t. Unilateral classical pure H.H. of medium degree, active for 4 years. **h** The panoramic view in centric occlusion discloses the typical radiographic picture of an unilateral pure H.H. of medium degree. **i, j** TMJ tomograms including the rami and angles show the unmistakable shape and structure of a H.H. deformity of the left side, in comparison with the normal right side. **k** The lateral cepha-



logram also demonstrates the remarkable difference between the two hemimandibles. **l** The mandible in p.a. projection in the closed position shows clearly the length difference between both sides of the face due to downward growth of both the maxilla and the mandible on the left side. **m** The mandible in p.a. projection at the maximum opening position discloses better than any other radiograph the difference in length of the halves of the mandible. **n** The measurements of the various parts of the mandible and the maxilla on the tracing of the panoramic view disclose the measureable differences between the normal and the pathological sides very clearly. **o** Diagrammatic illustration of the planned corrective surgery: only resection of the condyle and transposition of the surplus of the left inferior rim to the right side. **p, q** With the help of cast cap splints with training flanges on the normal side, mouth opening without deviation of the mandible is obtained. **r–t** Normal occlusion and normal position of the formerly tilted teeth after additional postsurgical orthodontics

crease in thickness of the ascending ramus and the condyle and its neck on the hyperplastic side. It also demonstrates the lateral rotation of the lower border and the angle of the "normal" right side. There is very little recognizable difference in height between the two sides of the maxilla. The radiograph of the hand and fingers showed that the epiphyseal plates of the fingers and those of the radius and ulna were almost completely closed, at the patient's age of 16 years.

The tracing of the panorama projection (Fig. 31n) permits measurements to diagnose the actual differences between the various sections of the two mandibular

halves and also the difference in height between the maxilla on the normal and pathological sides: The difference in length of the horizontal rami measures 5 mm, although the Pg-point is on the facial midline. So the difference is due to the increase in volume of the angle of the hyperplastic side. The trunks differ by 9 mm in length, obviously due to the increase in mass of the bone of the horizontal ramus and the contralateral decrease. The ascending rami differ by 15 mm in length, partially due to the additional increase in length of the trunk as well as of the articular process of the hyperplastic side. The difference between these two processes measures

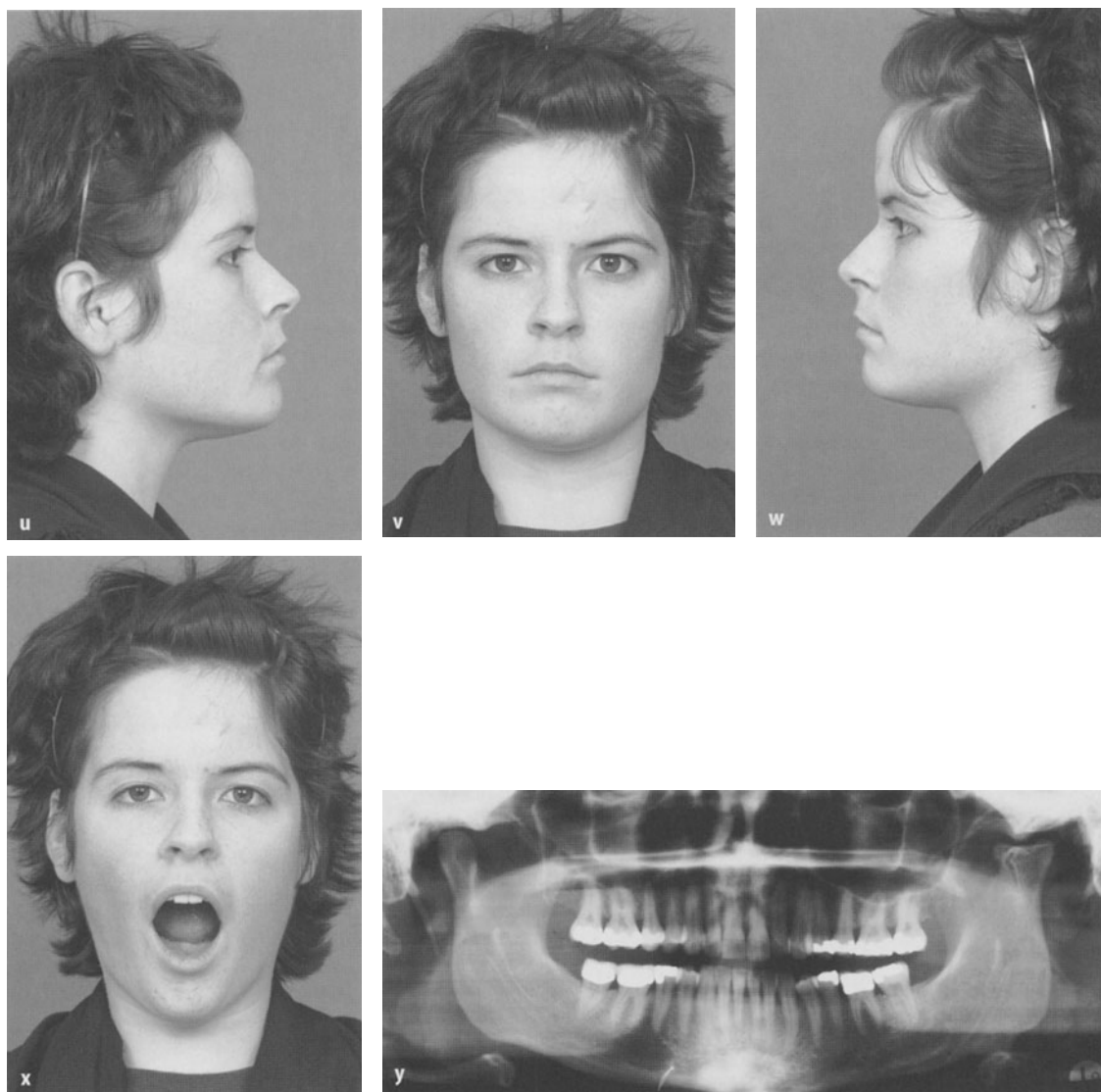


Fig. 31u–y. Unilateral classical pure H.H. of medium degree, active for 4 years. **u–x** Patient's final appearance is normal and so is the mouth opening, without deviation. **y** Panoramic view of the mandible nearly 16 years after surgery discloses that almost equal hemimandibles have developed. Instead of the condyle there is a broad flat surface on the top of the short neck

8 mm. Measurement also permits comparison of the width of the necks and the condyles themselves. The height increase of the left maxilla measures 2 mm more than the normal side.

Provisional Diagnosis. Typical unilateral pure H.H. of medium degree.

Treatment Plan. As follows:

1. Intracapsular high condylectomy
2. Freeing the left mandibular nerve
3. Transplantation of the surplus height of the left body to the lower border on the right side (Fig. 31o)
4. Postoperative use of a cast cap splint with training flanges in order to encourage mandibular opening and closing without deviation or occlusal disturbance.

It was not found necessary in this case to perform a Le Fort I osteotomy and a sagittal splitting osteotomy of the rami to rotate the lower half of the facial skeleton. The vertical asymmetry was not so severe that a very pleasing aesthetic and functional result could not be achieved without that additional amount of surgery.

Actual Treatment and Result. Surgery and postoperative treatment was carried out as planned. Four days after surgery British type cast cap splints, with training flanges on the right side, were cemented to the teeth. It permitted the patient immediate mouth opening and forced her to open and close the mouth without deviation to the resected side (Figs. 31p,q).

One year after surgery there was a perfect occlusion (Fig. 31r-t). The tilting of the teeth of the right side towards the affected side was corrected with the help of the patient's orthodontist (Dr. R. Schwitzer) who had kindly referred this interesting case. There was complete facial symmetry with an essentially unnoticeable reduction in height of the right side. And there was normal mouth opening without deviation to the side of resection of the condyle (Fig. 31u-x).

The occlusion and the patient's outward appearance were the same 15 years and 9 months after surgery but the panoramic view in central occlusion is very interesting (Fig. 31y): both sides of the mandible have become almost equal in shape and height. This is particularly true for the horizontal rami. Comparing the presurgical panoramic view with this discloses a very clear increase in vertical height of the right horizontal ramus. This is not only due to the transplanted inferior rim from the left side but also due to clear increase in height of the right alveolus by 5 mm in the first molar region. There is still coarse trabeculation visible in the structure of the left hemimandible, as compared with the normal side, in particular in the condylar neck area. There were no TMJ problems, in spite of the missing upper half of the

condyle. The surface of the high condyle resection has turned into a flat end of the thick neck, but without any functional impairment.

Histology. Micrographs of the resected condyle taken earlier showed that the articular surface was formed by soft tissue of completely normal appearance, comprising a distinct articular, proliferative, and hypertrophic layer. Abundant marrow spaces bordering directly on the cartilage and prominent cartilage remnants embedded in the subchondral bone indicated active endochondral ossification. Surprisingly, the appearance of the articular tissue as well as of the zone of erosion resembled that found in two cases of H.E. (Figs. 48u and 53m). However, unlike in the latter, the marrow spaces and trabeculae of the remainder of the spongiosa also appeared inconspicuous.

Diagnosis. Actively growing condyle of fairly normal microscopic structure.

Comment: Regrettably, only old photomicrographs could be examined, and these did not reveal any indication of condylar hyperplasia, such as signs of enhanced growth or the characteristic appearance of subchondral bone (Fig. 79a).

Discussion. This is a classical case of pure unilateral H.H. of medium degree. It shows all the classical physical signs of the abnormality, both clinically as well as radiologically. The 1 year and 15 year postoperative results prove clearly that a limited amount of surgery is sufficient for such a medium degree of H.H. This case also shows that a H.H. can cease even at the age of 15 years and 6 months.

The patient as well as the orthodontist should be informed presurgically about the surgeon's philosophy of treatment planning. In this case, the resection of the condyle did not result in a visible shortening of the increased height of the patient's affected side because there was occlusal contact between the upper and lower teeth and the maxilla was not raised. The symmetricalization between left height and right vertical shortness of the mandibular body was achieved only by the resection of the convex lower border of the left side and transplantation to the lower border on the right side. This would also have been necessary when a rotation procedure on the lower half of the facial skeleton was performed. Therefore rotation would not have saved the patient from equalization surgery of the height differences in the bodies.

The decision to perform a condylectomy was made for two reasons: the elimination of possible relapse caused by hyperactivity, as the patient was still very young, and secondly the possibility of obtaining histology information. The decision to perform condylectomy was not unwise, as we know that high condylectomy

does not cause any functional or pain problems, assuming there is proper postoperative physiotherapy.

Conclusion. There are classical cases of pure H.H. in which the abnormal growth may become obvious by the age of 11 years and already comes to a stop at the age of 14 years. Such a case can show all the classical clinical and radiological findings of that type of misregulation of growth due to the hyperactivity of the condylar M-

growth regulator. The type and amount of surgical correction depends very much on the degree of deformity and the surgeon's treatment philosophy. Since one cannot be certain that at the age of 15 $\frac{1}{2}$ years the condylar growth regulation factor will not become hyperactive again, intracapsular high condylar resection may seem to be indicated, particularly since such a high resection does not seem to have any functional or pain disadvantages.

Unilateral, Pure H.H., Active Over a Period of 20 Years (Fig. 32)

Sex and Age. Female, 40 years of age.

Aetiology and History. No pertinent family history, no trauma preceding the onset of the asymmetry. The patient produced photographs of herself taken at various ages. At the age of 14 there was no recognizable asymmetry. At the age of 20 the H.H. was well established. The patient had the impression that it was still increasing, but very slowly. She complained of TMJ pain, more on the side of the abnormality (left) than on the right side. She was referred for surgery because of her TMJ problems.

Clinical Findings. In the patient's facial asymmetry, the difference in length (vertical height) between left and right sides is rather impressive (Fig. 32a–c). The longer (higher) left side seems flatter with less contouring than the right side. The lower border on the left side is not only remarkably lower than normal but it is also slightly medially rotated. The lower border on the other side is more prominent laterally than is normal, in particular in the angle area where it looks almost like a mild degree of masseter hypertrophy. However, palpation of the right muscle in contraction as well as in relaxation rules out a diagnosis of masseter hypertrophy. The midline of the chin coincides with the facial midline, but the left half of the chin is much more prominent than the right. The lip commissure droops slightly towards the longer side of the face. There is a marked difference in the distance between the right corner of the mouth to the eye than that on the left side. The latter is remarkably longer. Sensory and motor nerve activity in the face seems normal. Mouth opening is not reduced but there is a slight deviation to the shorter side of the face. The occlusal plane is oblique, though not very severe, descending on the longer side (Fig. 32d). The teeth are in rather poor condition with a deep overbite in occlusion (Fig. 32e–g). The midlines of the upper and lower teeth are in the facial midline. There is no lingual tilting of the teeth noticeable on either side. – The chewing musculature is tender on palpation.

Radiographs. The orthopantomogram shows the typical H.H. deformity of the mandible (Fig. 32h), with enlargement of the left horizontal ramus and corresponding height reduction on the right side. Both condylar heads are large, but the right is within the range of normal while the left is significantly larger than the right. It has an almost normal shape but with an anterior "beak". The neck of that condyle is only slightly thicker than that on the right side and its length is also almost the same as the other side. – There is the typical, smooth, rounding of the angle area on the affected left side, with a rather large radius compared with the pronounced angle on the right side. Both sides have a clear cortical covering at the lower border, more on the normal right side than the H.H. side. There is a clear cortical covering all the way down the posterior border of the ascending ramus. The mandibular canal is situated rather far from the downward convexity of the lower border on the hyperplastic side, in contrast to findings in cases in which the hyperactivity of the condylar M-growth regulation factor causes a rapid increase in mandibular volume. The height of the right body is slightly reduced. The mandibular canal on the right side, in this case, is nearer to the slightly concave lower border than in a normal mandible, obviously due to the reduction in body height. The trabeculation seems only very slightly wider on the side of the H.H., but of similar thickness to the normal side. The enlarged condyle has a coarser trabeculation and shows some sclerosis.

The measurement of the tracing of the OPT (Fig. 32i) reveals that the left ascending ramus is 15 mm longer than the right, measured on the orthopantomogram from the upper surface of the condyle to the Ag-point. In both second molar regions there is a difference in body height of 13 mm and in the premolar regions of 5 mm. The distance between the inferior extension of the maxillary sinus and a line drawn between the two AH-points reveals a difference of 2 mm more on the H.H. side. The length of the trunks differs by 10 mm. The length of the articular processes differs by 1 mm

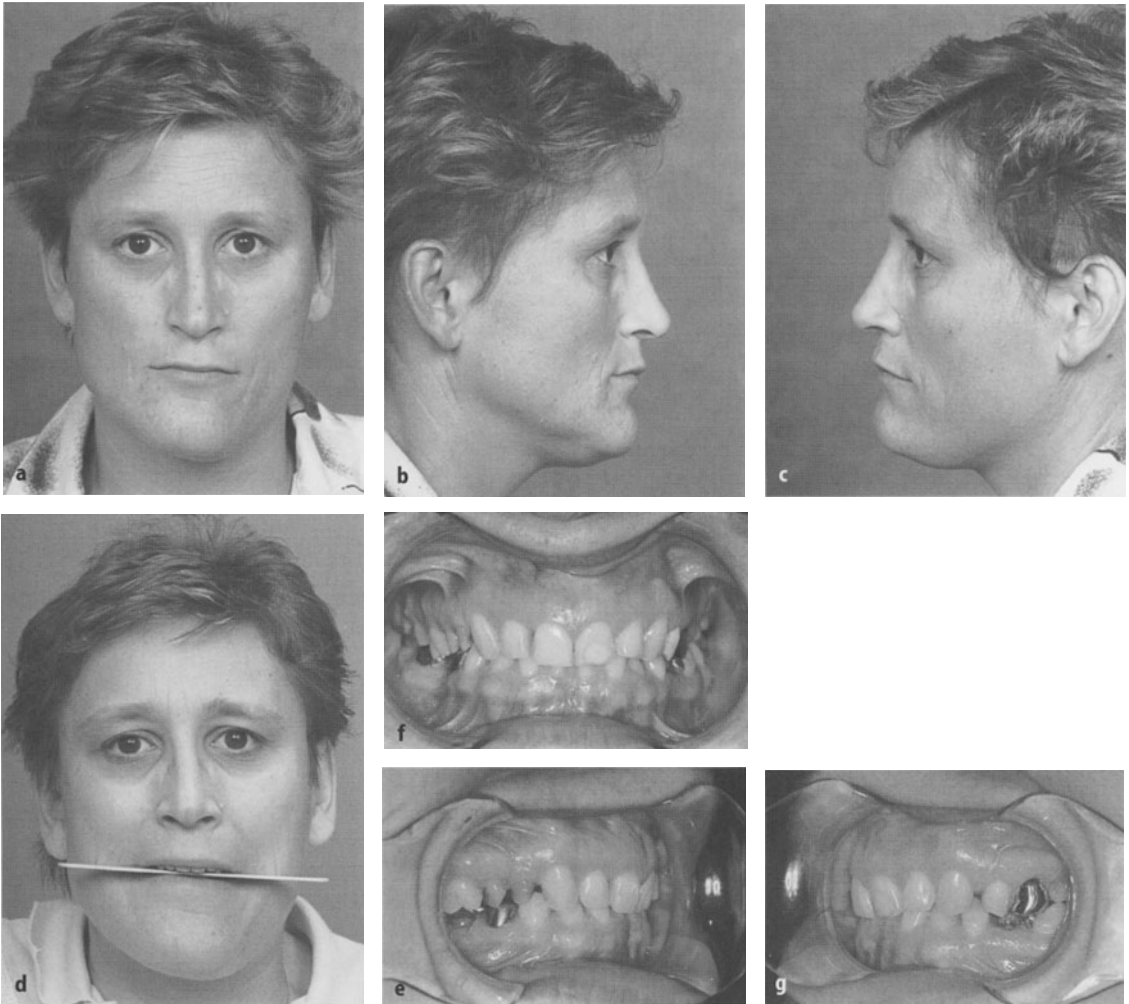


Fig. 32a–g. Unilateral pure H.H., active over a period of 20 years. **a–g** Patient's appearance and occlusion showing the difference to that of the patient in **Fig. 31**, due to the very slowly developing H.H. (over a period of 20 years)

only, but the width of the condyles differs by 6 mm in favour of the H.H. side.

TMJ tomograms and mandible p.a. projection at maximum mouth opening were unfortunately missing from the records.

The lateral cephalogram shows extremely well the height difference between the two sides and also the downward convexity on the H.H. side (Fig. 32j).

Scintigraphy. The investigation with technetium 99 resulted in an asymmetrical picture of the two TMJ regions. There was a difference of 17% more cellular activity on the H.H. side.

Provisional Diagnosis. Very slowly growing, over 20 years, typical unilateral H.H., still active at the age of 40 years.

Treatment Plan. As follows:

1. Intracapsular high resection of the condyle
2. Freeing of the left mandibular nerve
3. Resection of the left lower border by 10 mm and transfer of that piece of bone to the right lower border
4. Additional correction of chin asymmetry if necessary
5. No upwards repositioning of the maxilla
6. Postoperatively, use of cast cap splints with training flanges

Actual Treatment. The various surgical steps were carried out as planned by a surgeon in training. For the high condylectomy, a retrotragal incision was used to approach the TMJ. All other surgery on the mandible was performed via an oral approach. The cast cap splint

with training flanges was left in place for 10 weeks. The patient was followed for 7 years. The shape of the mandible was almost equal on both sides. There was clear corticalisation on the inferior borders and condyles of almost equal size on each side.

Histology. Macroscopically, the resected specimen was of more or less normal shape, although it appeared enlarged, measuring about 18 mm in medio-lateral diameter. Microscopically, the articular surface of the condyle was formed by fibrocartilage which, however, disclosed a clearly defined superficial, intermediate, and deep zone only in small areas. The major part of the cartilage revealed severe degenerative changes such as fraying of the articular surface (Fig. 32k, m) as well as vertical clefts and splits reaching down to the calcified cartilage (Fig. 32l). These signs of osteoarthritis were accompanied by indications of on-going regressive and, to a lesser extent, progressive cartilage remodeling. Thus, the tidemark, i.e., the uncalcified-calcified cartilage junction, followed an irregular course, calcified cartilage frequently formed spurs and tongues protruding

into the subchondral bone (Fig. 32m), and in the central aspect of the specimen, a prominent cavity filled with loose fibrous connective tissue and cartilage extended from the deep parts of the cartilage down into the subchondral bone (Fig. 32k) Signs of regular endochondral ossification were, however, absent.

Diagnosis. Osteoarthrotic condyle lacking signs of growth.

Comment: Apart from the increased size of the specimen, no histological signs indicated condylar hyperplasia. It can be inferred that active enlargement of the condyle had come to a standstill by the time of surgical intervention and that the enhanced activity revealed by scintigraphy was due to bone remodeling associated with osteoarthritis.

Discussion. Hyperactivity of the condylar growth regulators can be very mild, going on for over 20 years without producing a grossly exorbitant anomaly. This constant mild increase in mandibular volume permits the mandibular nerve canal and the maxilla to behave dif-

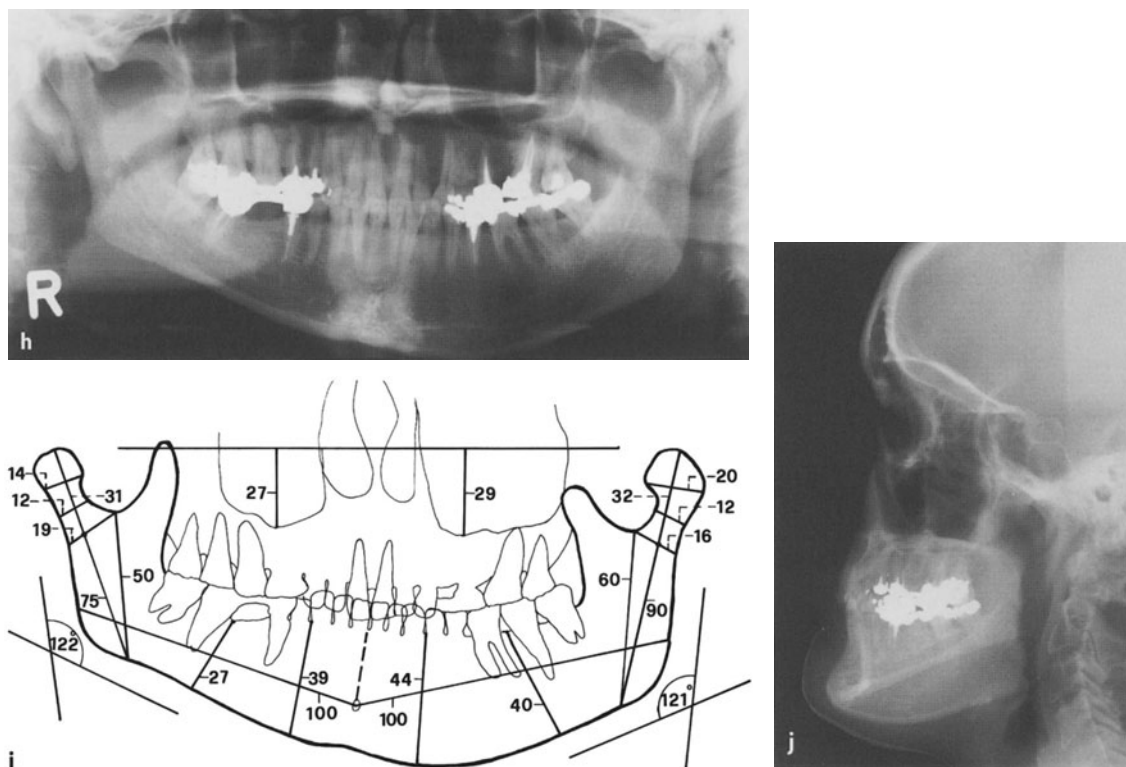


Fig. 32h-j. Unilateral pure H.H., active over a period of 20 years. **h** The panoramic view shows the classical picture of H.H., but with the mandibular canal clearly higher up on the affected side. **i** The tracing of the panoramic view discloses more clearly the difference between left and right. **j** The lateral cephalogram shows the typical findings of the unilateral H.H. with one horizontal ramus much more inferior than the other side. The reduction in height of the contralateral horizontal ramus seems more pronounced than on the panoramic view. This is due to its outward rotation

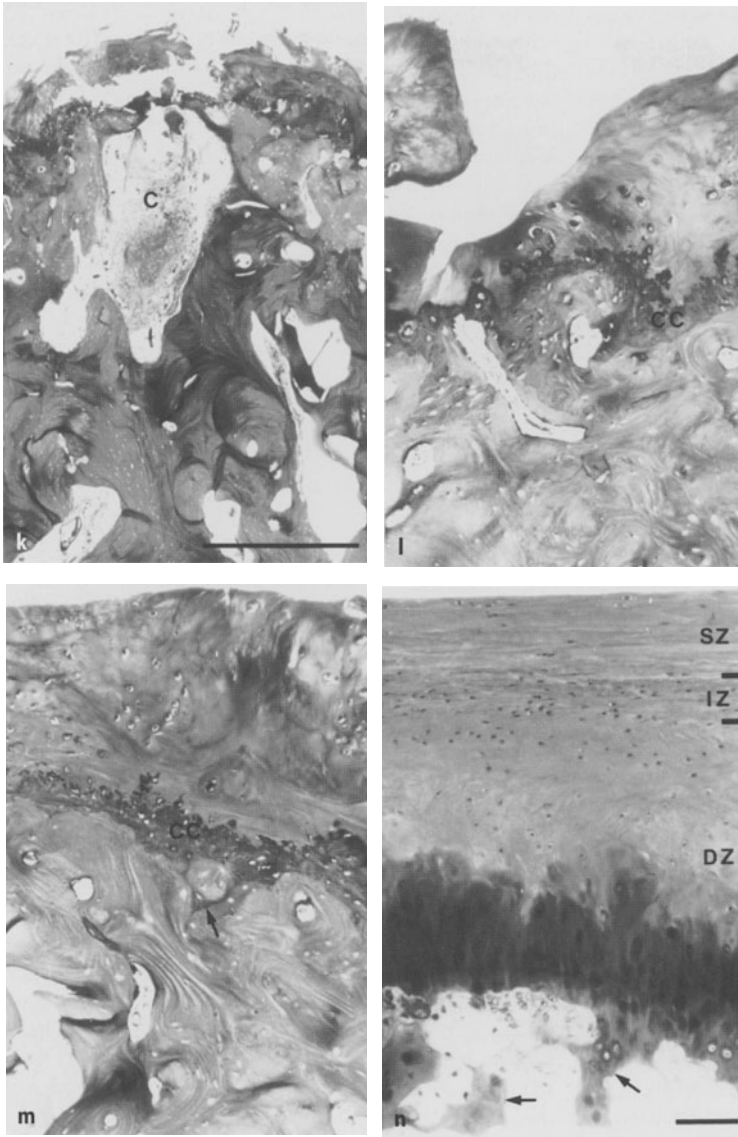


Fig. 32k–n. Unilateral pure H.H., active over a period of 20 years. Microscopic appearance of the articular tissue of the excised condyle (**k–m**) in comparison with that seen in a normal specimen from a female of about 44.5 years of age (**n**). Note the pseudocyst-like cavity **C** filled with loose connective tissue and cartilage **k** as well as the cleft reaching down to the calcified cartilage **CC** **l**. Where the articular surface is intact **m**, cells are missing from some areas and form clusters in other places. In the subchondral bone, a spicule of cartilage (*arrow*) indicates minor endochondral ossification. Similar signs of bone formation can also be found in a normal situation (**n arrows**), usually associated with conspicuous, heavily metachromatic cartilage in the deep zone **DZ** (**SZ** superficial zone, **IZ** intermediate zone. **k–m** Paraffin sections, **n** plastic section, toluidine blue, original magnification, **k** $\times 33$, *bar* = 1 mm, **l–n** $\times 130$, *bar* in **n** = 100 μ m)

ferently to rapidly developing hyperactivity cases. The trabecular structure remains fairly normal, the condyle does not become enlarged very much and irregular in shape. The downward growth of the maxilla is rather negligible and is not due to increase in alveolar height but rather due to the expansion of the maxillary sinus. The mandibular canal is not displaced to the lower border of the mandible as is always seen in the rapidly growing cases. This fact suggests that there is a real increase in mass of the alveolar bone in the rapidly growing cases, which seems to push the mandibular nerve down and the teeth up, causing the downward convexity of the lower border and the round angle with a considerably enlarged radius.

The fact that no histological signs of H.H. activity were found, but only signs of osteoarthritis, raises the question whether or not the enlargement of the condyle was the result of a long lasting chronic inflammatory process of the condyle only. And the second question will be, can chronic inflammation stimulate the growth regulators thereby producing one of the three condylar hyperactivity abnormalities? As we do not know any aetiology for the start of abnormal growth, slight inflammation in the condyle might be such a cause, but surely not the only one. Otherwise the mostly unilateral abnormal reaction and the often rather early beginning in childhood could not be explained and definitely not the start after growth has ceased. The possibility that ab-

normal enlargement of the condyle may initiate an arthrotic reaction is more likely.

Conclusion. When the hyperactivity of the M-growth regulator is very low grade, but goes on for many years, the form of the mandibular abnormality becomes the same as in an active case. As the hyperactivity goes on for over 20 years the trabecular structure of the increased alveolar bone, the ramus and the condyle re-

mains almost normal. The bone production does not seem to press the mandibular nerve down to the mandibular lower border but it increases in volume. Its trabecular structure does not show the hyperactive dense bone production that it does in a more active case. Measurement of the panoramic view of this particular case demonstrates clearly that H.H. produces enlargement of all sections of the affected mandibular side.

Unilateral, Pure H.H. with Very Little Increase in Mandibular Horizontal Ramus Height in Spite of Gross Length Difference Between the Ascending Rami (Fig. 33)

Sex and Age. Female, aged 23 years and 6 months.

Aetiology and History. At the age of 14 the patient had had an accident and received incorrect treatment (according to the patient). Then her facial asymmetry started to develop. That ended at about the age of 18. Nothing had been done since. She sought information about possible treatment.

Clinical Findings. The patient has a much longer right face than on the left which is shorter than normal (Figs. 33a–c). The right side is flat and the left well contoured. The nose is very oblique towards the right side but with a straight bony bridge. The lip commissure as well as the occlusal plane are oblique (Fig. 33d), descending towards the longer right side of the face. The right lower border of the mandible is curved slightly downwards and medially placed while the left is rotated outwards with a strong angle area. Mouth opening is unimpeded with very little deviation to the shorter left side (Fig. 33e).

The rather prominent chin seems to be almost in the facial midline when looking at the patient from in front. But when looking from below the chin prominence seems to be shifted to the left (Fig. 33f). For clear diagnostic purposes as well as for treatment planning one has to be sure whether or not the chin is out of the middle of the face. In order to be certain, I drew a horizontal line above the eyebrows with a pencil and held a linear scale perpendicular to it onto the middle of the forehead. Then the situation became clear. To be more precise, it is better to do that on a very enlarged front view photograph of the patient (Fig. 33g). As the view of the patient's face from below may lead to a wrong diagnosis, such an evaluation on an enlarged photograph of that view also can be misleading. The reason is, that when drawing the horizontal line above the eyebrows on the enlarged photograph of the view from below, that line, although appearing to be, may not be horizontal.

The occlusion reflects the abnormality so perfectly that the experienced clinician should be able to make the correct diagnosis almost without seeing the patient or his radiographs (Figs. 33h–j). The midline of the lower teeth almost coincides with that of the upper, but is slightly over to the hyperplastic side. The lower incisors are tilted towards that higher right side where the lateral teeth are vertical, contrary to those on the left side which are tilted inwards, producing an apparently lesser teeth height than on the right side.

The facial soft tissues, musculature and innervation were intact. There was no muscle hyper- or hypotrophy.

Radiographs. The panoramic view (Fig. 33k) impresses by the difference in vertical height of the two sides of the jaws, the right side is 13 mm longer measured from a horizontal line between the two AH-points down to the lower border in the second premolar region. There is a 7 mm height difference in the body, between its lower border and the roots of the first molars. The body on the smaller left side has very little height while the hyperplastic right side has only a slight height increase when compared with a normal case. There is a slight downward convexity in the molar-premolar region on that higher side. The right angle is rounded, although not with a large radius. Its cortical covering on the lower border is rather thin compared with the normal while that on the non-hyperplastic left side is strikingly thick in spite of the remarkably reduced body height. The symphysis is in the facial midline. The left condyle and neck are normal in size and shape while the right condyle is clearly enlarged and rounded. – The trabecular structure of the right body is wider than that on the left side and its mandibular canal runs right along the lower cortical plate. – The occlusal plane is very oblique, tilting towards the longer (higher) side.

The tracing of the panoramic view (Fig. 33l) permits exact evaluation of the amount and the location of the differences between the two mandibular halves.

The TMJ tomograms showed the left condyle and angle to be of normal size and shape. The right was clearly enlarged with a rounded anterior “beak”. It seemed to have a fine, almost translucent trabecular structure.

The mandible p.a. projection at maximum mouth opening (Fig. 33m) discloses impressively the height difference between the shorter left and the longer right ascending rami. It also shows the typical H.H. inward

rotation of the body and ascending ramus of the longer side and the outward rotation of the normal left side, with no deviation of the chin towards either side. The left condyle, of normal shape and size, has an inferiorly directed fine trabeculation. The condyle on the H.H. side is rounded and moderately enlarged and has a clearly coarse and more irregular trabeculation. The maxilla shows a rather strange anatomy. The nasal septum is severely bent to the left non-hyperplastic side and the right maxillary sinus is quite a bit larger than the left. The right side of the maxilla is situated more in-



Fig. 33a–f. Unilateral pure H.H. with very little increase in mandibular horizontal ramus height in spite of gross length differences between the ascending rami. **a–f** Patient's appearance with all the typical signs of unilateral pure H.H.: rotation of the lower half of the face, oblique occlusal plane and lip commissure, pseudo-deviation of the chin prominence to the non-hyperplastic side and gross difference in vertical facial height of the two sides

feriorly and seems to be displaced to the right side. However, the midline of the upper teeth is in a vertical line with the crista galli. The medial displacement of the hyperplastic right mandible simulates a displacement of the maxilla to the right side.

Provisional Diagnosis. Unilateral pure H.H. with little increase in the body height in spite of the existence of severe facial asymmetry.

Treatment Plan. High condylectomy on the right side; rotation of the lower half of the facial skeleton via a Le Fort I-type osteotomy and a bilateral sagittal splitting procedure on the rami; resection of the surplus of the right lower border, using it as an onlay graft to increase the left mandibular body height. For that, the mandibular nerve must first be freed.

Actual Treatment. None. The patient was from outside Switzerland. She sought information only.

Discussion. Although this case showed about the same severe facial asymmetry as did the former case, the radiological picture was quite different. There was about the same height difference between the H.H. side and the normal side due mainly to vertical increase in the ascending ramus, but only to a minor degree in the body. The vertical increase in the ascending ramus was followed by severe downward growth of the maxilla while in the former case the maxilla remained almost in a horizontal position. There arose the question whether the accident during puberty had pushed the maxilla downwards and caused the right condyle to commence an M-regulator-induced hyperactivity. This was a question I was unable to answer. It was conspicuous that in this case there was such a small increase in the body height and no division of the chin prominence into two eminences, which we normally find in every case of unilateral H.H. There was also a remarkably reduced height of the contralateral mandible and maxilla which contributed to such a severe facial asymmetry.

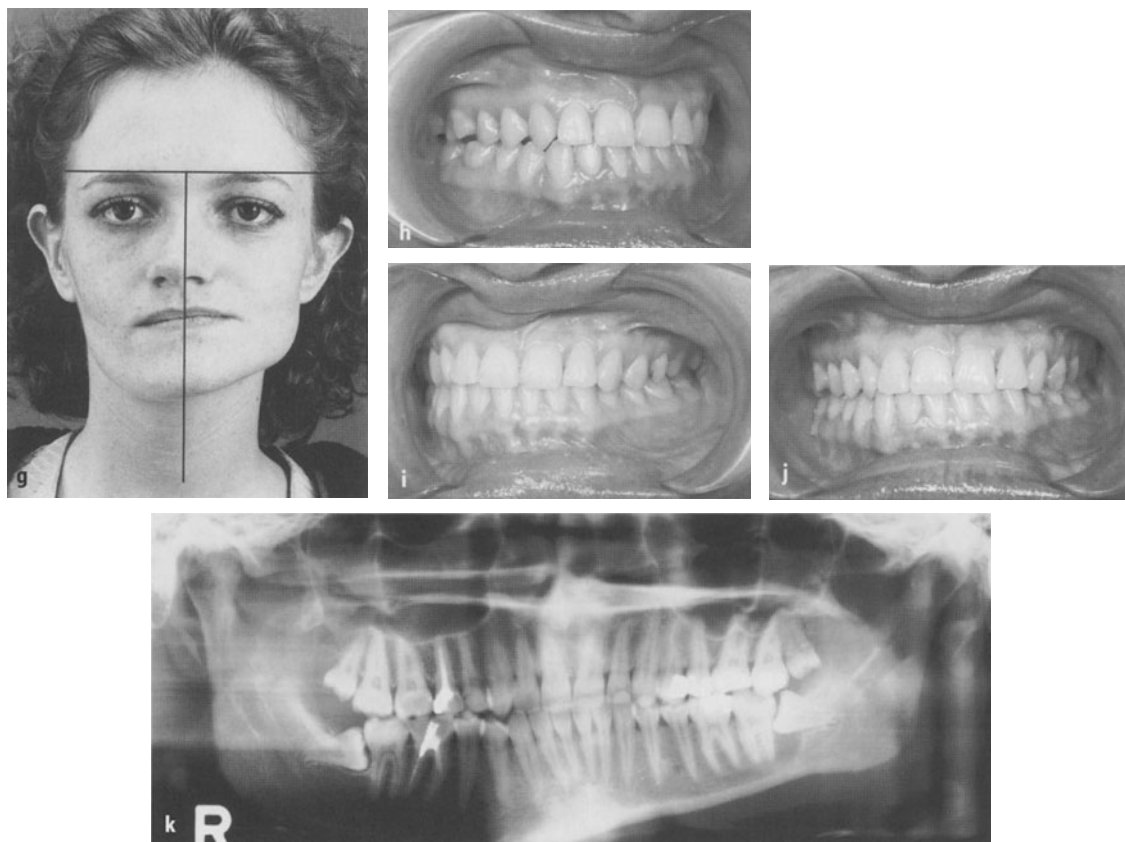


Fig. 33g–k. Unilateral pure H.H. with very little increase in mandibular horizontal ramus height in spite of gross length differences between the ascending rami. **g** The symmetry evaluation of the face in the frontal view proves that the chin prominence is not out of the facial midline, but the nose is severely out. **h–j** The patient's occlusion is very typical of an unilateral H.H. case. **k** The panoramic view in the closed position discloses clearly the very remarkable height differences between the left and right sides of the face, due to the mandible as well as the maxilla on the right side and reduction of the horizontal ramus height on the left side

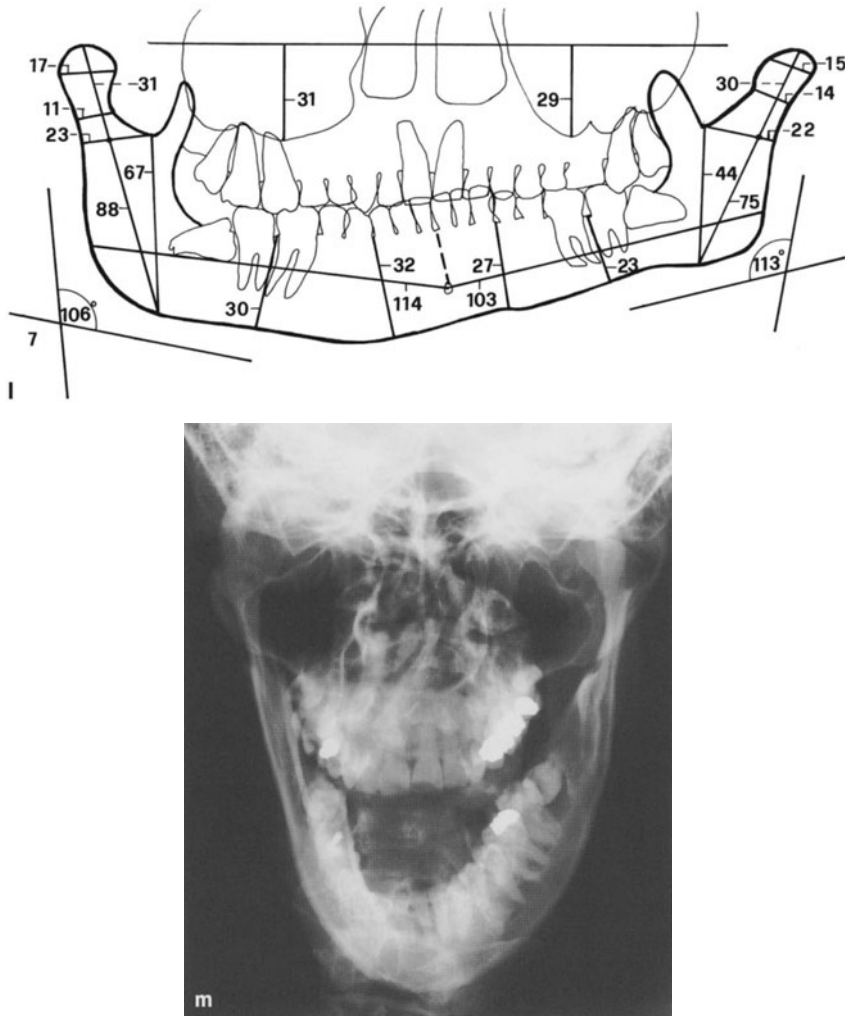


Fig. 331, m. Unilateral pure H.H. with very little increase in mandibular horizontal ramus height in spite of gross length differences between the ascending rami. **l** The tracing of the panoramic view with its measurements discloses the amount and location of the differences between the two sides. **m** The mandible p.a. projection at maximum opening shows the typical shape of the mandible in an unilateral H.H. case and some abnormalities in the maxilla, most probably due to a fracture in childhood

The inexperienced might have diagnosed this case as a hybrid form of H.H. and H.E. as the chin prominence seemed to be quite a bit further over to the opposite side than the tilted tip of the nose. But if one were ever in doubt what was in, and what was out, of the facial mid-line, then the following principle has to be applied: *“When you are not sure whether a facial asymmetry exists or not, place the patient’s head in the upright position, then draw a horizontal line above the eyebrows and an imaginary line perpendicular to it from the middle of the forehead. Then it can be seen clearly whether a facial asymmetry exists or not and what is on which side of the face, and, what the differences are between the two sides of the face”* (Principle No. 16).

Conclusion. There are cases with clear signs of pure unilateral H.H. which differ in their skeletal background from other such H.H. cases, although the outward appearance may be very much the same. Whether this was due to an accident during puberty in this case or not is difficult to decide. The diagnosis of H.H. is confirmed by the clear enlargement of the condyle and the height increase in the body and its downwards convexity.

Unilateral H.H. with a Tumour-like Enlargement of the Upper Half of the Condyle (Fig. 34)

Sex and Age. Male, 44 years of age.

Aetiology and History. During the past 2 years, the 44-year-old patient had noticed that his right side of the face had become longer than the left side and an open bite had developed on the right side. He did not know any possible cause for this. He had the impression that there was now no further abnormal growth. He wanted information about any possible treatment.

Clinical Findings. The facial appearance (Fig. 34a) is typical of a moderate degree of H.H. The affected right side is longer than the other side. The lower border has a downward convexity and is tilted medially. The cheek is flat. The other side is rather short in height with the lower border of the mandible slightly outwardly rotated. The chin prominence is exactly in the facial midline with its right half further down and a bit more prominent than the left. The lip commissure is oblique, with the right corner remarkably lower than the opposite side. Mouth opening is not restricted, but with some deviation to the left side.

The dentition is poor (Fig. 34b). There is a diastema between the upper central incisors. It is displaced to the left side of the facial midline, which is congruent with the axis of the right upper incisor. The midline of the lower front teeth seems to be shifted to the higher right facial side by the width of 1 1/2 lower incisors, compared with the middle of the upper diastema. But since the facial midline runs through the axis of the right upper incisor, the actual shift of the lower midline to the right side is one-half the width of a first lower central incisor only and that is mainly due to the tilting of the lower front teeth to the higher facial side. Although the occlusal picture might suggest that the mandible is shifted remarkably to the side of the H.H., this is not the case. The facial midline indicates that the teeth on the right side remain vertical while the left teeth, in particular the lower ones, are tilted lingually. In spite of the rather poor tooth situation we can still clearly recognize the typical sign of the H.H. anomaly in the occlusal picture: the teeth on the affected side seem to be longer than those on the normal side. But there is an open bite, increasing from the right canines posteriorly.

Radiographs. The panoramic view (Fig. 34c) shows a very interesting asymmetrical shape of the mandible. The two sides are entirely different. The right ascending ramus is much higher and wider than the left. There is clear enlargement of its condyle for which the upper half is mainly responsible. The angle is rounded and the body on the same side is remarkably higher than on the

left and shows an inferior convexity, mainly in the premolar region. The mandibular canal runs well down to the inferior border. The left half has a gracile ascending ramus and condyle of normal height, and a somewhat stretched angle. Its body height is clearly reduced in the molar region. The maxilla seems to be in a horizontal position.

The TMJ-ramus tomograms (Figs. 34d, e) permit clear visualization of the tumour-like enlargement of the upper half of the right condyle with a clearly uniform trabecular structure. The neck is not elongated nor thickened. The left condyle, neck and ascending ramus are within the normal range in size, shape and length.

The measurement of the tracing of the panoramic view indicates very clearly the differences between the two hemimandibles (Fig. 34f). Every length measurement is noticeably greater on the (right) H.H. side.

Provisional Diagnosis. Unilateral H.H. due to tumour-like enlargement of the upper half of the right condyle.

Treatment Plan. High condylectomy on the affected side and additional sagittal splitting of the same ascending ramus if the open bite does not completely close after the high condylectomy. For height symmetrization of the mandibular bodies, either the right lower border should be transferred to the left, or a piece of lyophilized cartilage should be fixed transorally to the left lower border.

Actual Treatment. Regrettably the patient did not agree to any surgery.

Discussion and Conclusion. This case was diagnostically interesting in three ways. The tumour-like enlargement of the upper half of the affected condyle was probably a product of the M-growth regulator. It was obviously a part of the condyle itself. It did not show any demarcation towards the normal bony structures as one would have expected to find in the case of a benign tumour even of bony origin. The enlargement was therefore very interesting for histological examination. It was a pity, that the patient refused surgery. The second point was that the abnormal position of the diastema between the upper central incisors, together with the gracile shape of the left hemimandible with its stretched angle could easily have led to a misdiagnosis of additional left H.E.

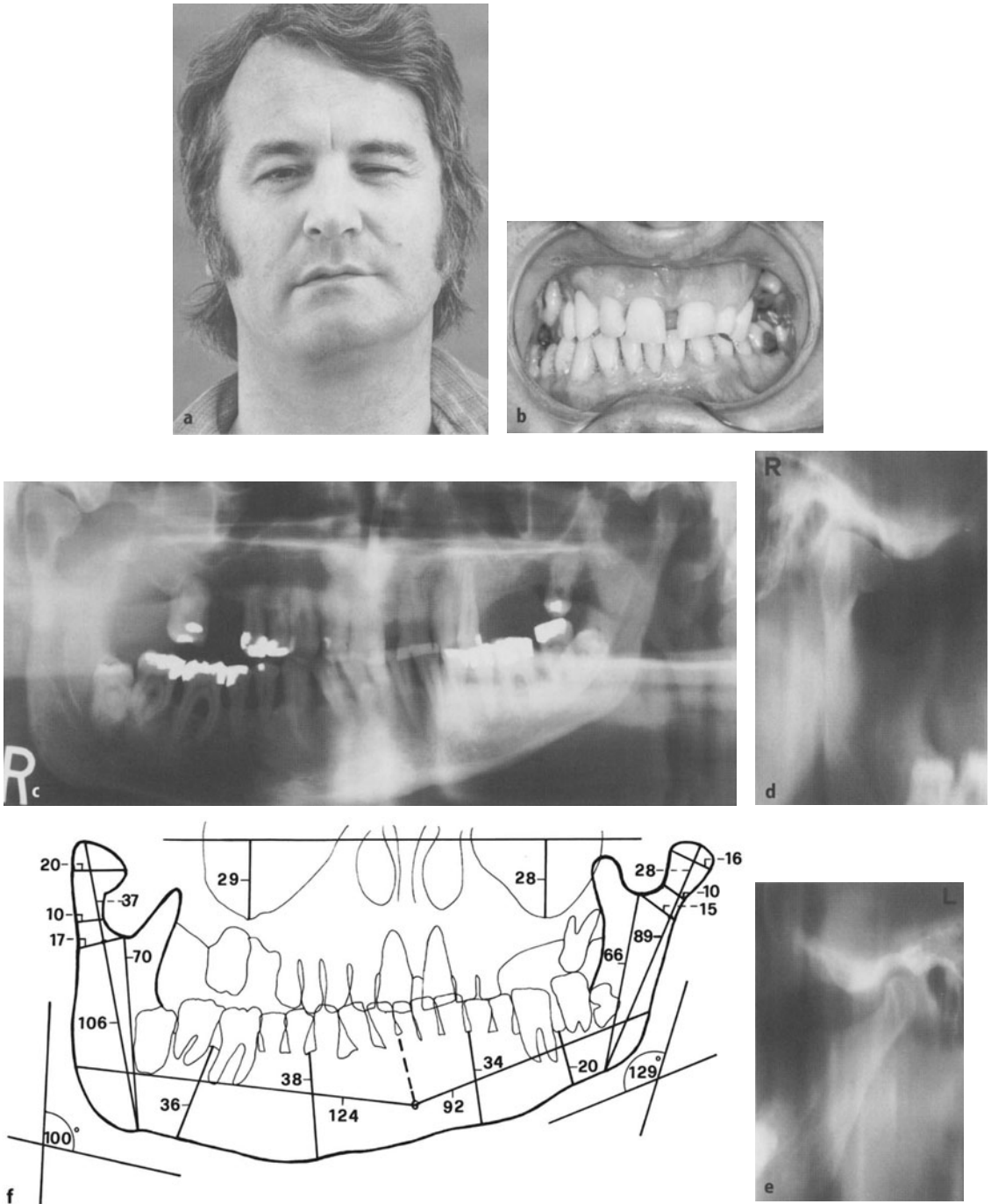


Fig. 34a–f. Unilateral H.H. with a tumour-like enlargement of the upper half of the condyle, which started at age 42. **a** Patient's appearance in front view showing the typical facial disfigurement due to H.H. of the right side. **b** The occlusal photograph does not show a very clear situation due to the poor dentition except that the teeth on the side of the H.H. seem longer than those on the other side. **c** The panoramic view is very interesting as it raises the question whether or not the unilateral elongation of the right ascending ramus is due to a tumour of the condyle or not. **d, e** The TMJ ramus tomograms do not answer that question clearly, except for the fact that the enlargement of the right condyle would not permit a clear diagnosis of the type of tumour. **f** The measurement evaluations of the tracing of the panoramic radiograph eliminate all doubts

The fact that there is an open bite on the affected side might suggest that this is due to a benign tumour of the condyle, as normally observed in such a case. But the inferior bowing of the lower border of the affected mandible and the increase in its alveolar height are not normally seen in cases of benign condylar tumours. The open bite is easily explainable by the fact that the abnormal mandibular growth on the right started only 2 years ago. As the patient is 44 years old, one does not expect that the maxilla would follow the mandible downwards as normal growth has ceased and at that age, the alveolus with its teeth will not follow so rapidly.

Unilateral, Rudimentary H.H. (Fig. 35)

Sex and Age. Male, 20 years old.

Aetiology and History. Patient came for consultation because he had noticed a slight facial asymmetry. He could not remember any cause for it, nor when it had started. He believed that it had not changed during the past 6 months.

Clinical Findings. The right side of the patient's face is slightly longer than the left side (Fig. 35a,b). There is also asymmetry of the prominence of the chin with the right side a little lower than the left side. But there is no deviation of the chin prominence to either side. The right mandibular lower border is slightly lower than the left. The right cheek is a bit more voluminous than the other side. Palpation reveals this to be of muscular origin. The left half of the face seems slightly hypoplastic, in particular the zygoma-infraorbital rim area. The lip commissure is parallel to the eye level, but with the left corner of the lip further back than on the right. There was normal function of the facial sensory and motor nerves and mandibular opening was normal.

In occlusion there is an open bite on the right side and a class I occlusion on the left side (Fig. 35c-e). The lower front teeth are slightly tilted towards the right, and the lower midline is shifted over to the right by the width of half a lower incisor. The left cheek teeth seem shorter than the right ones when viewed from the front.

Radiographs. The panoramic view (Fig. 35f) shows a minor difference between the left and right mandibles. Both condyles and their necks seem almost equal in size and shape. However, the right ascending ramus is clearly longer than the left. The right angle has the typical rounding compared with the normal angle form on the left side. There is a noticeable increase in the distance between the apices of the right molars and bicusps and the lower border of the mandible when compared with the left side which is within the range of normal. The overall slight increase in size of the right mandible

Although we do not have any histological or scintigraphic proof of a H.H. in this case, I have no doubt that the deformity of this mandible is due to a H.H. growth abnormality. The type of enlargement of the condyle and its neck, the additional height increase of the trunk of the ramus, the rounding of the angle, the inferior bowing of the lower border, the height increase of the alveolar bone and its coarse trabeculation, are all signs clearly recognizable by measurements of the tracing of the panoramic view. They would not be found in a case of condyle enlargement due to a benign tumour.

ends at the middle of the chin. There is no reduction in height of the left body compared with normal. On both sides, the mandibular canal runs at its usual distance from the lower border. The lower front teeth are slightly tilted to the right side and the midline is also over to the right by the width of half an incisor. There is a height difference between left and right maxillae, the left being about 4 mm higher in relation to a line drawn between the upper border of the two external auditory meati. The trabeculations of the mandible are within normal on both sides.

TMJ tomograms including ramus (Fig. 35g, h) show clearly a somewhat larger right condyle and longer ascending ramus than on the left side. This is also true for the mandibular body in the molar region.

The *mandible p.a. projection maximum opening* (Fig. 35i) shows an almost symmetrical mandible and yet with clear findings of rudimentary H.H. on the right side: the right condyle is larger than the normal left one. The right ascending ramus is longer and seems narrower because of the outward rotation of the left side. The right side of the prominence of the chin is wider than that on the left. This projection also discloses a clear hypoplasia of the right side of the maxilla: the floor of the nose is higher than on the left side and the right alveolar-zygomatic crest is clearly shorter than that on the contralateral side as well as the whole right maxilla.

The result of measuring the mandible on the tracing of the panoramic radiograph also helps to make the correct diagnosis of a rather rudimentary form of H.H. This is clearly recognizable in the tracing in this case (Fig. 35j).

Skeletal scintigraphy with 500 MBq Tc-99m-DPD revealed an asymmetrical uptake, slightly greater in the right condyle.

Provisional Diagnosis. Very rudimentary form of a still slightly active H.H. on the right side with a medium de-

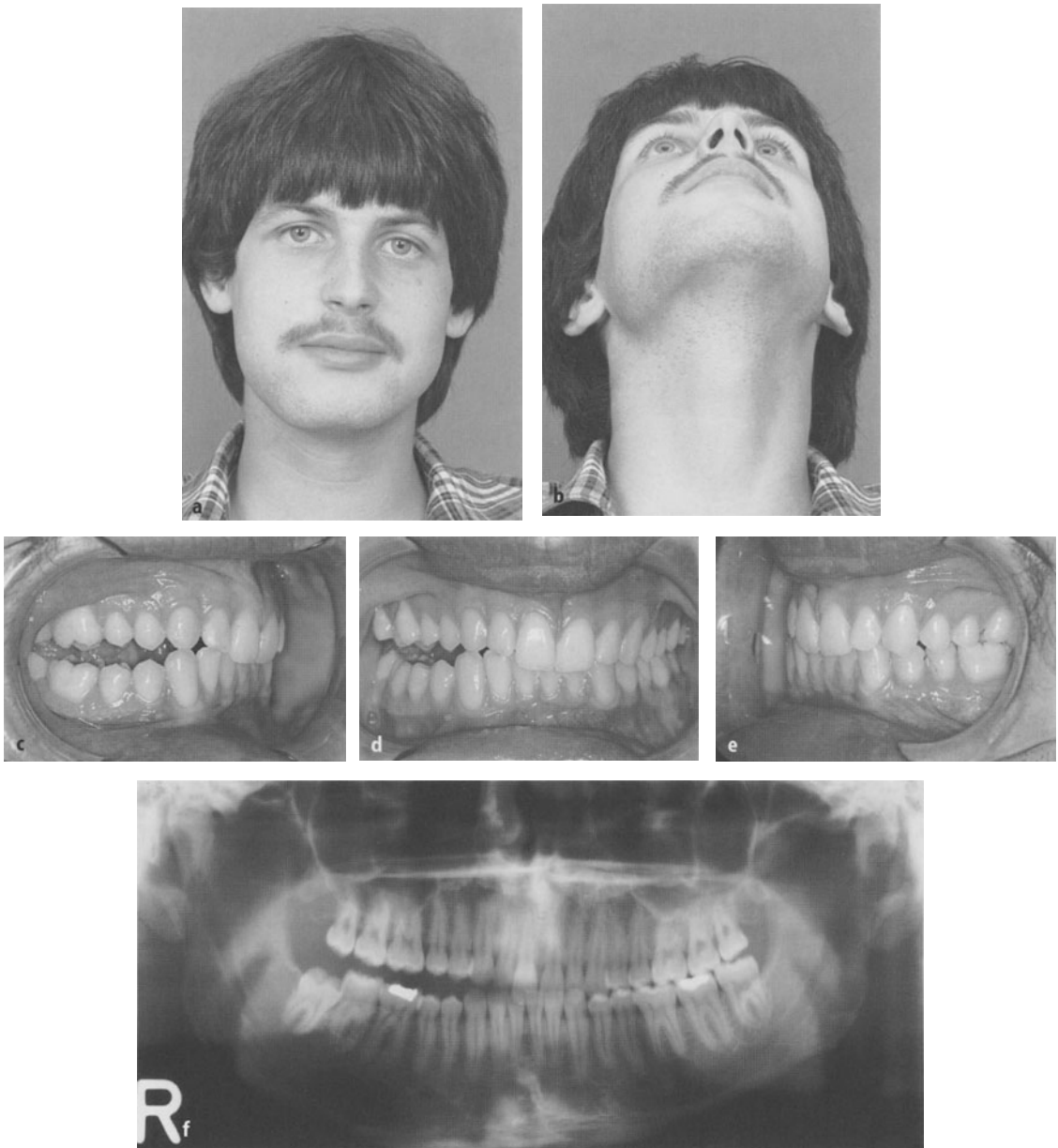


Fig. 35a–f. Unilateral rudimentary H.H. Patient's appearance in the **a** front view and **b** from below show a clear difference between the two sides of the face which does not automatically permit a diagnosis of an unilateral H.H. **c–e** The occlusal picture shows an open bite on the right side and a tilting of the lower front teeth towards the same side and the left cheek teeth seem shorter than the right ones. **f** The panoramic view permits the diagnosis of a right side rudimentary H.H., but the open bite on the same side does not seem to fit in with that diagnosis

gree of masseter hypertrophy on the same side. Hypoplasia of the right side of the maxilla with some lack of downward growth. In addition there is a medium degree of hypoplasia of the left zygoma and infraorbital rim.

Treatment Plan. A high condylectomy should be performed on the right side, thereby arresting further growth and the open bite may be partially closed. However, in such a case of insignificant, still on-going hyperactivity one could easily wait until it burns itself out in

order to avoid operating on the joint. Apart from the condyle problem, the maxilla must be rotated inferiorly on the right side and the left zygoma and left infraorbital rim need some augmentation. In addition, the masseter hypertrophy on the right side as well as the inferior border of the mandible and the chin of the same side need volume reduction for better symmetrization of the face.

Actual Treatment. The patient refused any kind of surgery; however, 1 year later he came back with no further change in asymmetry or occlusion, but no change of mind regarding treatment.

Discussion. In this very slow-growing, rudimentary type of unilateral H.H., the striking feature was the open bite on the side of the longer facial side. The question whether this was only due to the still slightly active H.H. or also only due to hypoplasia of the maxilla on the same side was answered by the radiographs and clinical

findings. On palpation, it seemed that the maxilla on the right was clearly hypoplastic compared with the left. This was confirmed by the p.a. projection of the mandible (Fig. 35i).

The hypoplasia of the maxilla on the H.H. side cannot be a consequence of the H.H. The contrary should be the case. There is no answer in the patient's history to the question of the cause of the vertical shortness of the right maxilla. As the floor of the nose on the right side is also higher up than that on the normal left side, I assume that the right side hypoplasia of the maxilla must be the consequence of damage to that side of the maxilla in childhood. As there was no surgery or radiotherapy in early childhood we will have to assume that it is due to an early trauma. The result means that the open bite situation on the rudimentary H.H. side is not due to fast mandibular downward growth but due to lack of normal maxillary growth development.

Regardless of the open bite which is well-known in very rapidly-growing hyperactive H.H. cases, there were

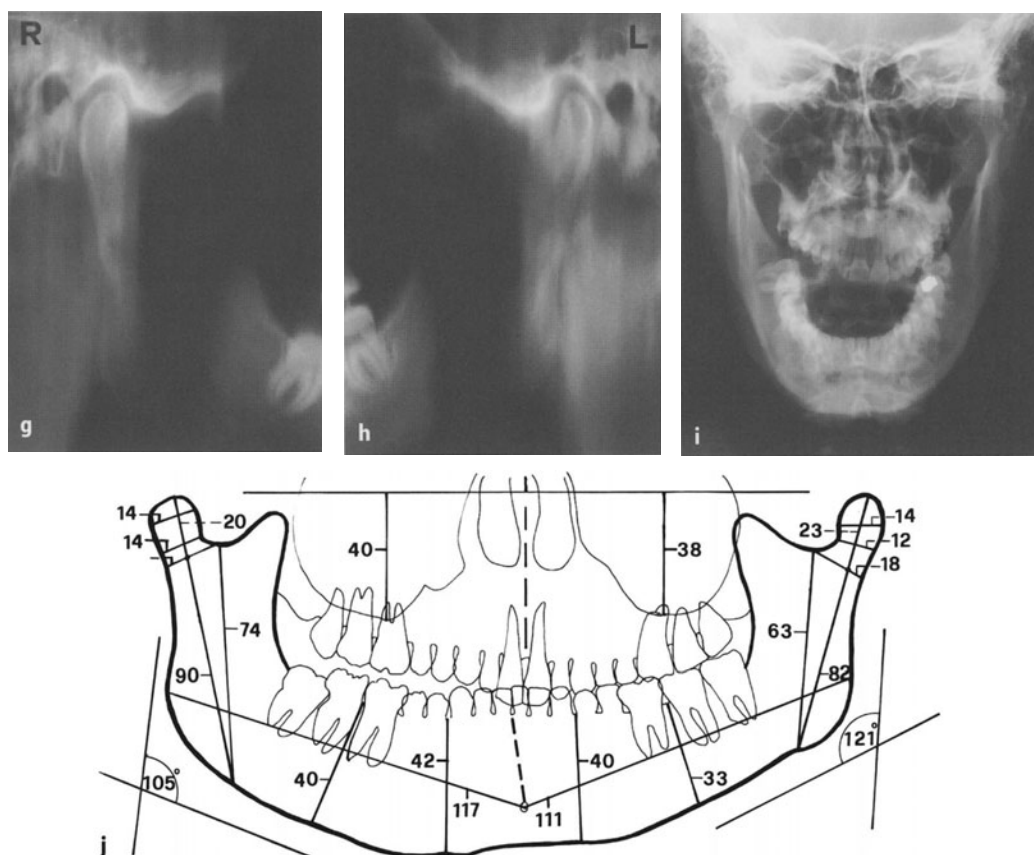


Fig. 35g-j. g, h The findings on the TMJ ramus tomographs are congruent with a rudimentary right side H.H. i The p.a. mandible projection on maximum opening confirms the rudimentary H.H. on the right side, but also answers the question as to the aetiology of the open bite on the same side. j The measurement evaluations of the tracing of the panoramic view confirm the diagnosis of a rudimentary H.H. on the right side and explains the existence of the open bite on the same side

all the classical signs of a rudimentary H.H., in the shape and size of the condyle and the ascending ramus and body of the affected half of the mandible.

The lip commissure did not tilt downwards on the H.H. side as one would have expected. Normally, we find the corner on the H.H. side lower than that on the normal side. In this case it was higher because of the deficiency of downward growth of the right maxilla as a result of its hypoplasia. That suggests that the vertical elongation of the ascending ramus and the inferior convexity of the body did not influence the position of the lip corner all that much in such a rudimentary case, as did the lack of downward growth of the maxilla.

The result of the scintigraphy also supports the diagnosis of a rudimentary hyperactivity on the right side. That hyperactivity cannot be due to still ongoing normal growth. In that case the other side would also have to be slightly active.

Conclusion. There are very rudimentary forms of H.H. which can appear together with an open bite of maxillary as well as mandibular origin. It is normally expect-

ed that the maxilla would follow the downward growth of the mandible during growth. Since it did not do so, the open bite in this case will be of mostly maxillary origin.

Additionally it must be mentioned that routinely, a p.a. projection of the mandible in the open and preferably also in the closed positions should be taken. In this case this projection shows the hemimaxillary hypoplasia of the H.H. side very clearly. An additional sinus view projection would show the maxillary situation even better. But the best projections for that problem would be CT-tomograms.

Another point we learned from this case: the position of the corner of the mouth on the affected side depends not only on the downward growth of the mandible but also on the growth (or lack of it) of the maxilla and the possible influence of the soft tissue volume.

Normally in the H.H. cases and in the hybrid forms, we expect a flatness of the cheek on the same side and a greater cheek volume on the normal side. In this case, the contrary was true because of a slight unilateral masseter hypertrophy.

Unilateral, Very Rudimentary H.H. and Contralateral Masseter Hypertrophy (Fig. 36)

Sex and Age. Male, 28 years of age.

Aetiology and History. The patient could not remember any possible cause for his facial asymmetry. He wanted to know why it had occurred. There was nothing similar in his family history. He said the left side had always been like this and that there was nothing wrong with his right side. He wished to know the cause of the swelling of his left angle area.

Clinical Findings. Apart from a rather prominent left angle area there is nothing else causing obvious facial asymmetry (Fig. 36a). The chin prominence is exactly in the facial midline. Its right side is a little more prominent than the left. The right cheek is flatter than the left and the right mandible is straighter compared with the left which tends to be turned outwards more than the right. The right lower border is slightly more inferior and its angle is more rounded than the left. The left side voluminous angle area is clearly partially due to a surplus of masseter musculature and its underlying bony protruberance. The mouth opening is normal without any deviation (Fig. 36b).

The full dentition in occlusion (Fig. 36c–e) shows the typical appearance of a mild H.H. abnormality. The upper and lower midlines almost coincide, but with a clearly noticeable tilting of the lower incisors to the right side. Due to a lingual tilting of the lower left lateral teeth they seem shorter in height than the right ones

which are clearly in a vertical position. The interocclusal relationship of the teeth on the right side is normal and on the left side there is a class II occlusion by the width of half a bicuspid. The occlusal plane is slightly oblique, tilting downwards to the right side.

Radiographs. In the panoramic view (Fig. 36f) the two halves of the mandible look very similar when not evaluated carefully. But there is a clear, although slight difference in the length of the ascending rami (12 mm) and bodies and also clearly in the size and shape of the condyles. These parts of the right mandible are larger than the normal left ones. The right angle is more rounded than the left with a rather large radius of its rounding. The right has much less of an irregular protuberance than the left and also much less of an antegonial notch. The right condyle is clearly larger than the normal sized left one. The right horizontal ramus is a little higher in the premolar region than normal and has there a mild inferior convexity. There is no real difference in the trabeculation; the mandibular canals on both sides seem to be at a normal distance from the lower border, the right moreso than the left. The maxillary sinus is slightly lower on the right side than on the left. There is no open bite.

TMJ-ramus tomograms (Figs. 36g, h): The left condyle and neck are of normal shape and size. The right

condyle is clearly enlarged, although not very strikingly so and it has a short but broad neck and a strong anterior or “beak”.

There is a striking difference between the two glenoid fossae. The left is rather narrow and there is an unusual articular prominence. The right glenoid fossa

seems considerably enlarged, mainly due to loss of the articular prominence. It seems to be widely abraded by the condyle’s anterior “beak”.

The mandible in *p.a.* projection in the open position (Fig. 36i) shows the typical picture of an unilateral H.H. and a rather normal contralateral side. The right hyper-

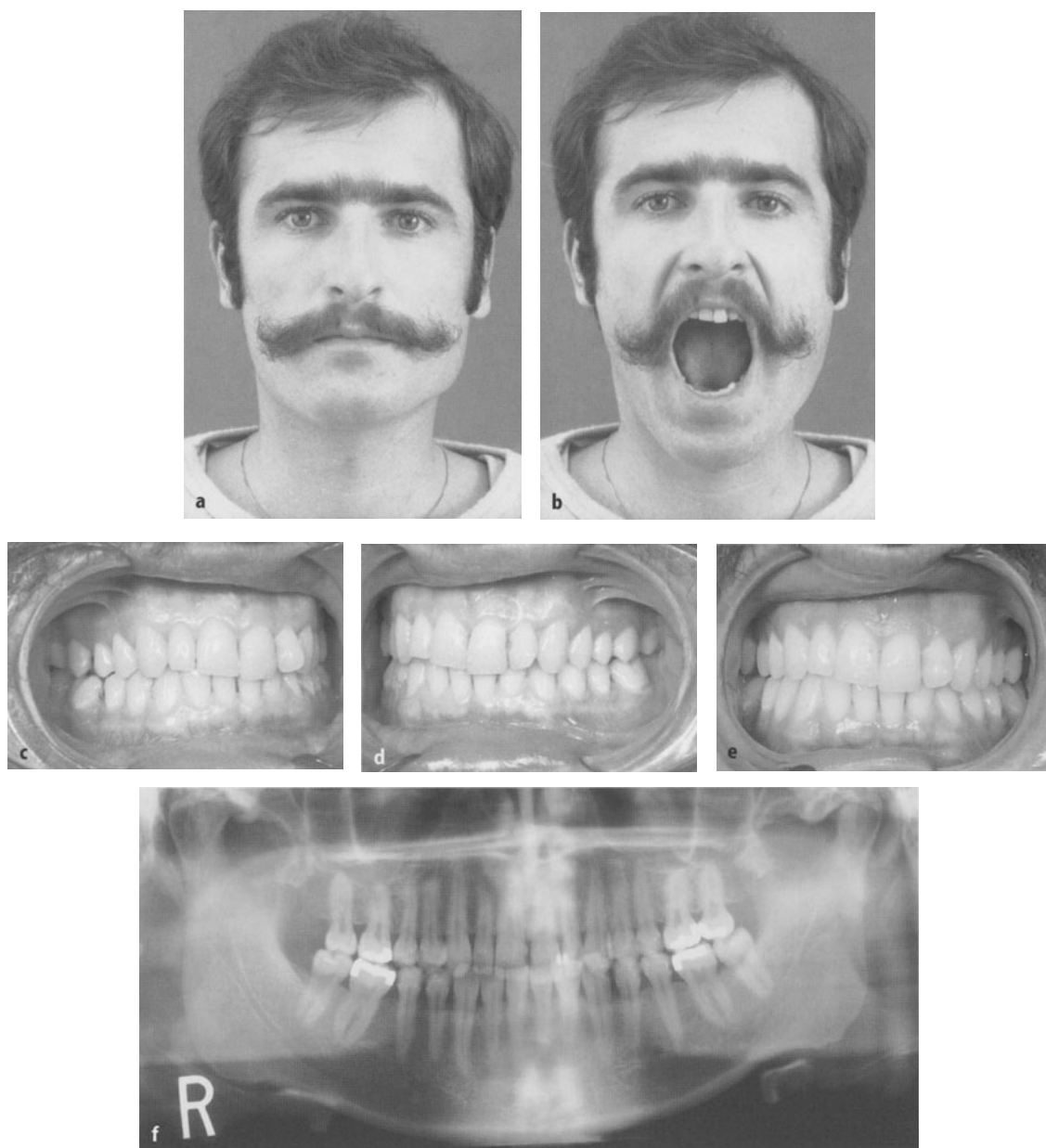


Fig. 36a–f. Unilateral, very rudimentary H.H. and contralateral masseter hypertrophy. The patient’s appearance in the **a** front view and **b** at maximum mouth opening, suggesting a surplus of contour and tissues in the left angle region. **c–e** The patient’s occlusion with “longer” teeth on the right side than on the left, typical of a H.H. on the right side. **f** The panoramic view seems to show very little differences between right and left sides except for the large condyle and the rounding of the angle on the right side

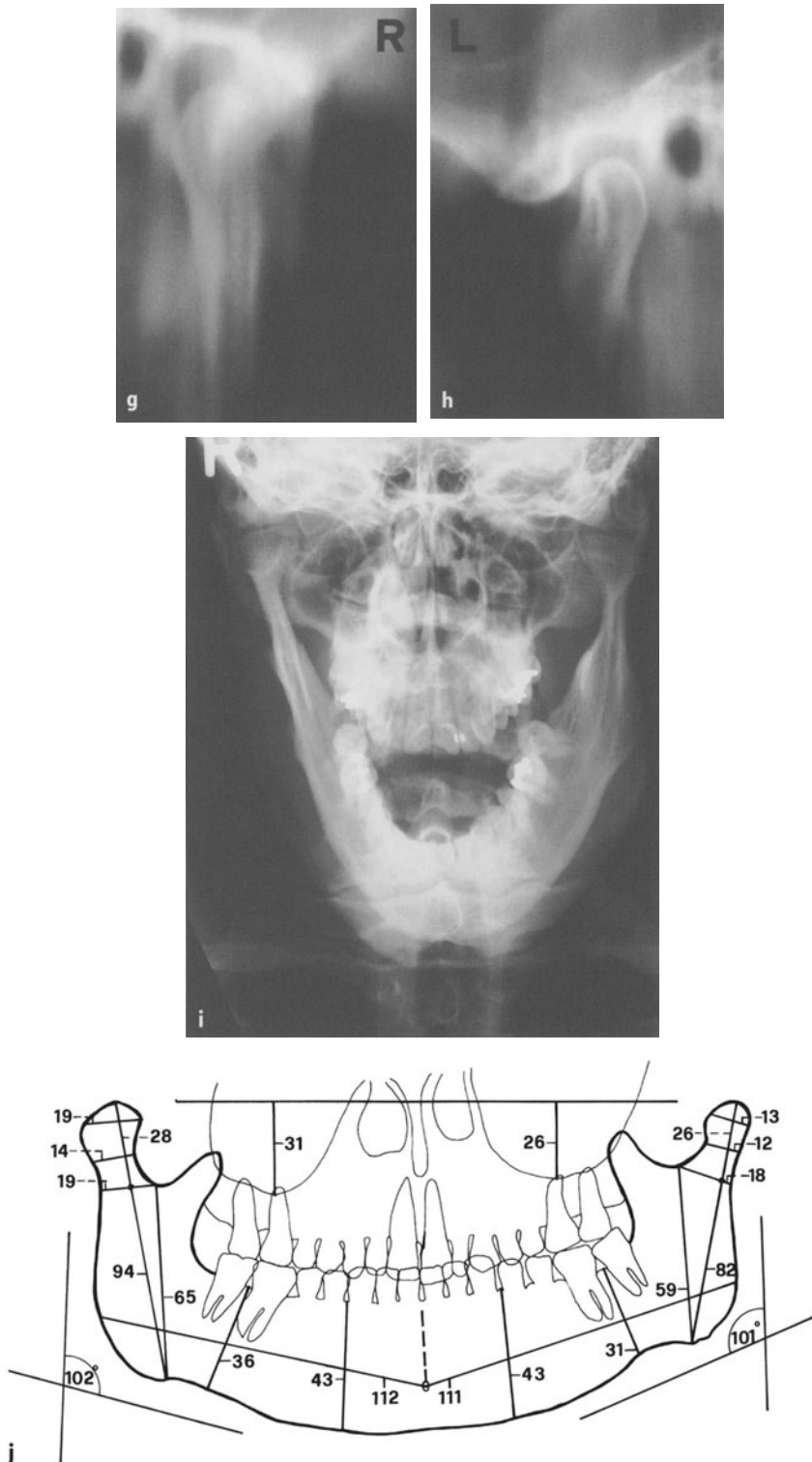


Fig. 36g-j. Unilateral, very rudimentary H.H. and contralateral masseter hypertrophy. **g, h** The TMJ tomograms show a clear difference in size between the two condyles and the glenoid fossae. **i** The mandible p.a. projection in the open mouth position shows the classical picture of a right side H.H. **j** The measurement results of the tracing of the panoramic view are significant for a H.H.

plastic side looks straight and slim and has a much longer ramus-condyle region than the other side which appears outwardly rotated as usual. The right condyle has a coarser trabeculation than the left. The left angle seems rather prominent.

The lateral cephalogram discloses that one side of the mandible extends further inferiorly than the other. It is clearly the right one with the more rounded angle than the left shorter one.

The tracing of the panoramic radiograph and its measurements made the diagnosis clear (Fig. 36j). There is a difference of 12 mm in length between the two ascending rami, 2 mm of it due to the difference between the two articular processes. The same is true for their width. The length of the two horizontal rami differs very little (1 mm), but their height in the molar regions differs by 5 mm. The maxilla on the right side is lower than that on the left. The inferior extension of the maxillary sinuses differs by 5 mm. Although the patient had no idea that something on the left side of his mandible was wrong, it could not be overlooked on this tracing of the panoramic radiograph.

Provisional Diagnosis. Very rudimentary H.H. on the right side and well-pronounced masseter hypertrophy on the left side.

Treatment Plan. Intracapsular high resection of the right condyle (for histology) and typical transoral partial resection of the left masseter muscle and angle.

Actual Treatment. The surgery was carried out by one of the trainees in the department in accordance with the plan.

Histology. Macroscopically, the resected specimen was of approximately normal shape and medio-lateral dimension (the antero-posterior dimension could not be assessed). Microscopically, bone in the entire articulating region of the condyle was covered by a transitional type of cartilage. In some areas, this cartilage resembled the fibrocartilage typical of adult specimens, while in other areas it appeared similar to hypertrophic growth cartilage (Fig. 36k). A distinct cell layer, analogous to the proliferative layer of growing condyles, constituted the intermediate zone, and the superficial zone, although damaged in isolated small areas, appeared similar to the articular layer. In the central aspect of the specimen, prominent collagen fibre bundles formed arcades around hypertrophic chondrocytes of the deep zone (Fig. 36k). In these areas, unusually numerous and large subchondral marrow spaces bordered directly on the cartilage, and the trabeculae of the spongiosa, incon-

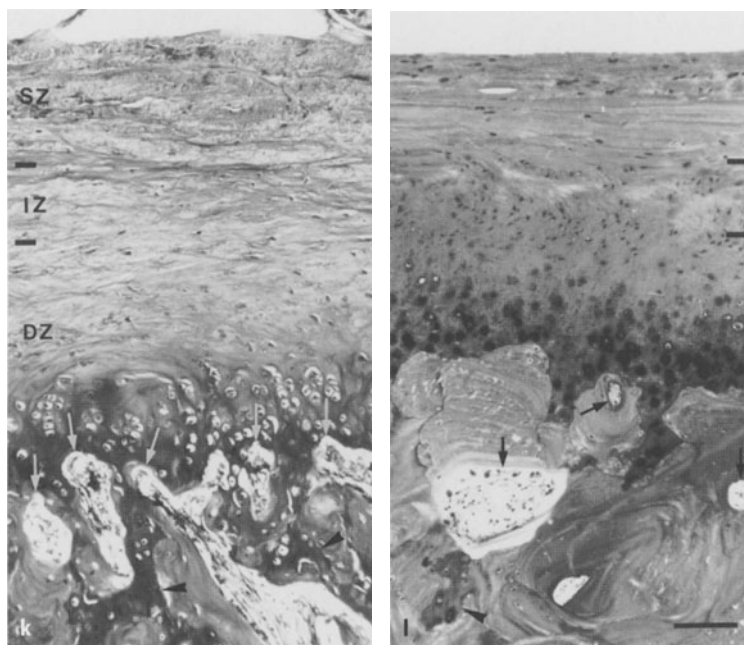


Fig. 36k, l. Unilateral, very rudimentary H.H. and contralateral masseter hypertrophy. Microscopic appearance of the articular tissue of the excised condyle **k** in comparison with that seen in a normal specimen from a male of about 26 years of age **l**. Note in the affected condyle **k** the hypertrophic cartilage occupying the deep zone **DZ**, numerous marrow spaces bordering directly on cartilage (*arrows*) and large cartilage islands in the subchondral bone (*arrow-heads*). These features contrast with the transitional type of cartilage, isolated marrow spaces surrounded by bone (*arrows*) and few subchondral cartilage rests (*arrow-heads*) in the normal specimen **l**. (*IZ* intermediate zone, *SZ* superficial zone; **k** paraffin section, **l** plastic section, toluidine blue, original magnification $\times 130$, *bar* = 100 μ m)

spicuous regarding thickness and arrangement (see Fig. 79j), contained cartilage remnants down to a depth of about 1.5 mm.

Diagnosis. Microscopically fairly inconspicuous condyle exhibiting residual growth.

Comment: Considering that this case was classified as a very rudimentary form of H.H., the inconspicuous appearance of the resected specimen is not surprising. Still, the observed signs of endochondral ossification clearly exceed those seen in age-matched control subjects, suggesting that condylar growth relative to the patient's age had been abnormal.

Discussion. Although the patient was referred by his dentist for correction of his left masseter hypertrophy, the routine clinical and radiological investigation made it clear that in addition a contralateral minor degree of H.H. existed. The left side clearly had a muscle and bone surplus in the angle region. Probably also due to the ex-

istence of a minor degree of surplus bone in the right angle – often found in cases of unilateral masseter hypertrophy – and due to the minor degree of H.H. on that side, the right angle did not show the typical rounding but still exhibited a certain degree of flat antegonial concavity.

The clearly existing antegonial notch in front of the left angle can be explained by the fact that the angle was enlarged due to the existence of a masseter hypertrophy on that side. Normally one would expect a condylar hypoplasia to be the cause for such an antegonial notch. But no condylar hyperplasia in this case existed.

The histological findings seem to confirm the existence of a very mild H.H.-hyperactivity.

Conclusion. It is easy to overlook a minor degree of H.H. if, on the contralateral side, another volume-producing deformity exists. Precise clinical investigation and clear evaluation of the radiographs is mandatory in all cases which one is seeing for the first time.

Unilateral, Juvenile H.H. and Late, Contralateral H.E. (Fig. 37)

Sex and Age. Male, 12 years and 4 months.

Aetiology and History. The boy's father was a general medical practitioner. He reported that from birth the boy's chin was not in the midline (no photograph available). During growth the facial asymmetry had gradually become more evident. It had resulted in a clear over development of the right and an under development of the boy's left side of the face. A disturbed occlusion existed on the right side even in the deciduous dentition. This was the father's actual description of the development of the anomaly. He had no explanation for the origin of the anomaly and no photographic proof of the situation at birth.

Clinical Findings. The 12-year-old boy is in very good general health. His appearance reveals the classical physical findings of pure unilateral H.H. (Fig. 37a–e): The right side of his face is much longer than the left. His right cheek is flat and that flatness extends to the lower border of the mandible and the lower border extends much more medially than normal. It has a clear downward convexity and ends exactly at the middle of the chin, which is precisely in the midline of the face. The left side seems less high than normal with the lower border of the mandible outwardly rotated. There is an oblique lip commissure, dropping from high on the left to low on the right. Mouth opening is unimpeded but it deviates to the non-H.H. side. Facial nerves display a normal reaction.

The occlusion (Fig. 37f–h) is twisted with a slight inward rotation on the left and an outward and downward rotation of the hyperplasia side. The lower front teeth are slightly tilted towards the right side and the cheek teeth on the normal side are in a slight maxillary cross-bite position. The midline of the lower front teeth is over to the H.H. side by the width of half a lower incisor, only partially due to their tilting.

Radiographs. The orthopantomogram (OPT) reveals the classical radiological picture of a H.H. on the right side (Fig. 37i): all sections of the right mandible are enlarged, the condyle and its neck, the height of the whole ascending ramus and also of the horizontal ramus. There is the typical rounding of the angle with a larger radius than normal and besides the typical increase in height of the horizontal ramus there is also an inferior convexity. There is only a very thin cortical covering recognizable in the molar and bicuspid regions. There is not only an increase in the height of the horizontal ramus but also in the width of its trabeculation, in the whole right mandible. The trabeculae themselves are coarse compared with the other side, both in the body as well as in the ascending ramus, condyle and its neck. The mandibular canal seems to be displaced down to the thin cortical covering at the lower border.

The body of the contralateral side is clearly reduced in height compared with normal. The symphysis is ex-

actly in the facial midline, but the front teeth are severely tilted towards the side of the hyperplasia. The upper incisor midline is in one line with the nasal septum. The midline of the lower front teeth is shifted towards the

hyperplastic right side by the width of half an incisor, mainly due to their tilting. There is space between the teeth on that side, probably due to an early extraction of the first molar, although nothing had been mentioned

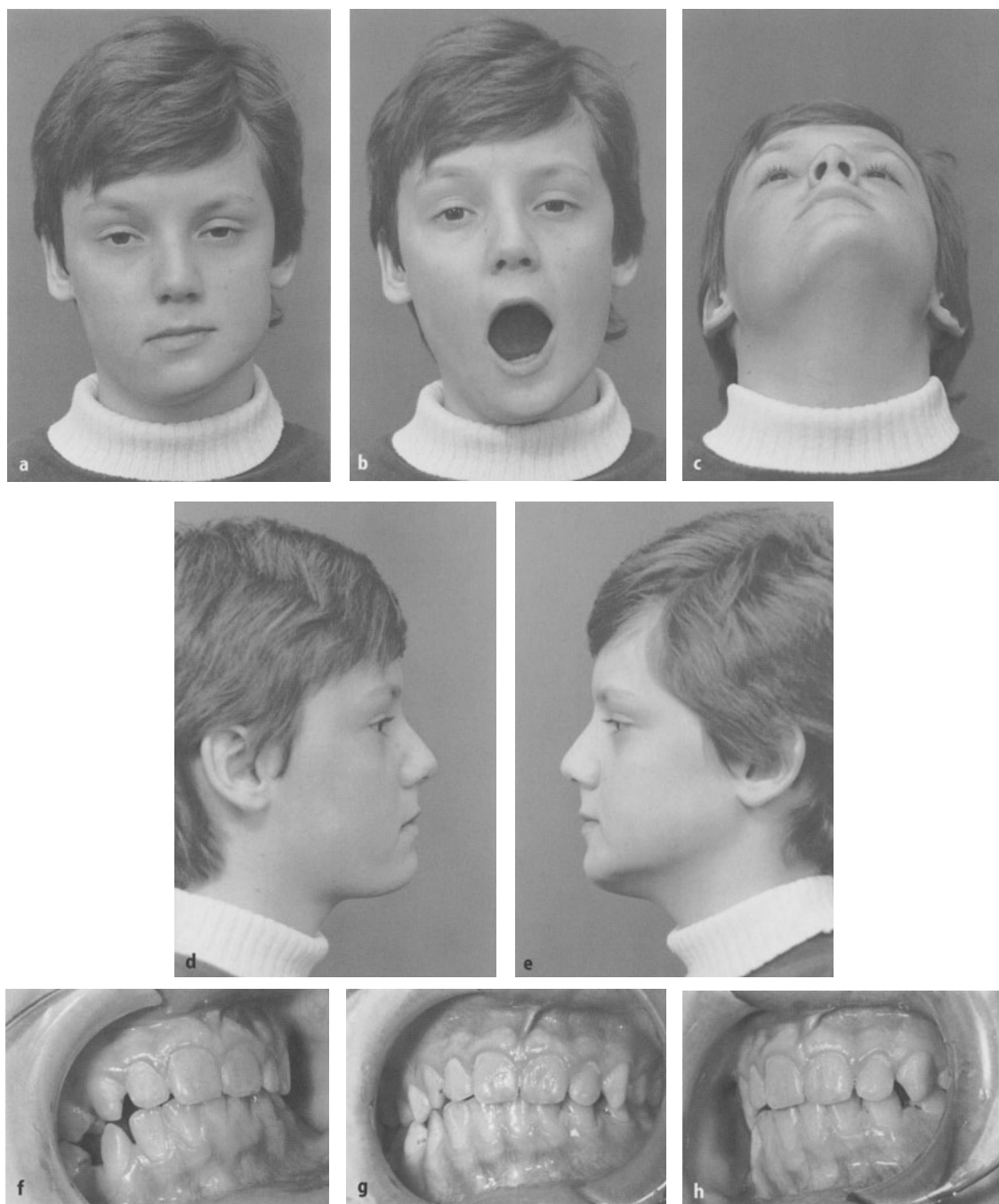


Fig. 37a–h. Unilateral juvenile H.H. and late contralateral H.E. **a–e** Typical appearance of 12-year-old boy with pure unilateral H.H. **f–h** Typical occlusion picture of unilateral H.H. **i** Classical panoramic view appearance of an unilateral right side H.H., at age 12

about this. The midline of the apices of the lower front teeth is inclined slightly to the right side, obviously only due to their tilting.

The right side of the maxilla is a few millimetres lower than the left. This is not due to an increase in alveolar bone but due to extension of the maxillary sinus.

The TMJ tomograms (Fig. 37j, k) show clearly the difference in size and shape of the condyles. The left side is normal, the right condyle is enlarged but not very deformed. It has a rather flat surface with a beak-like shape at its anterior part. The glenoid fossa is also rather flat as compared with normal and the other side. Al-

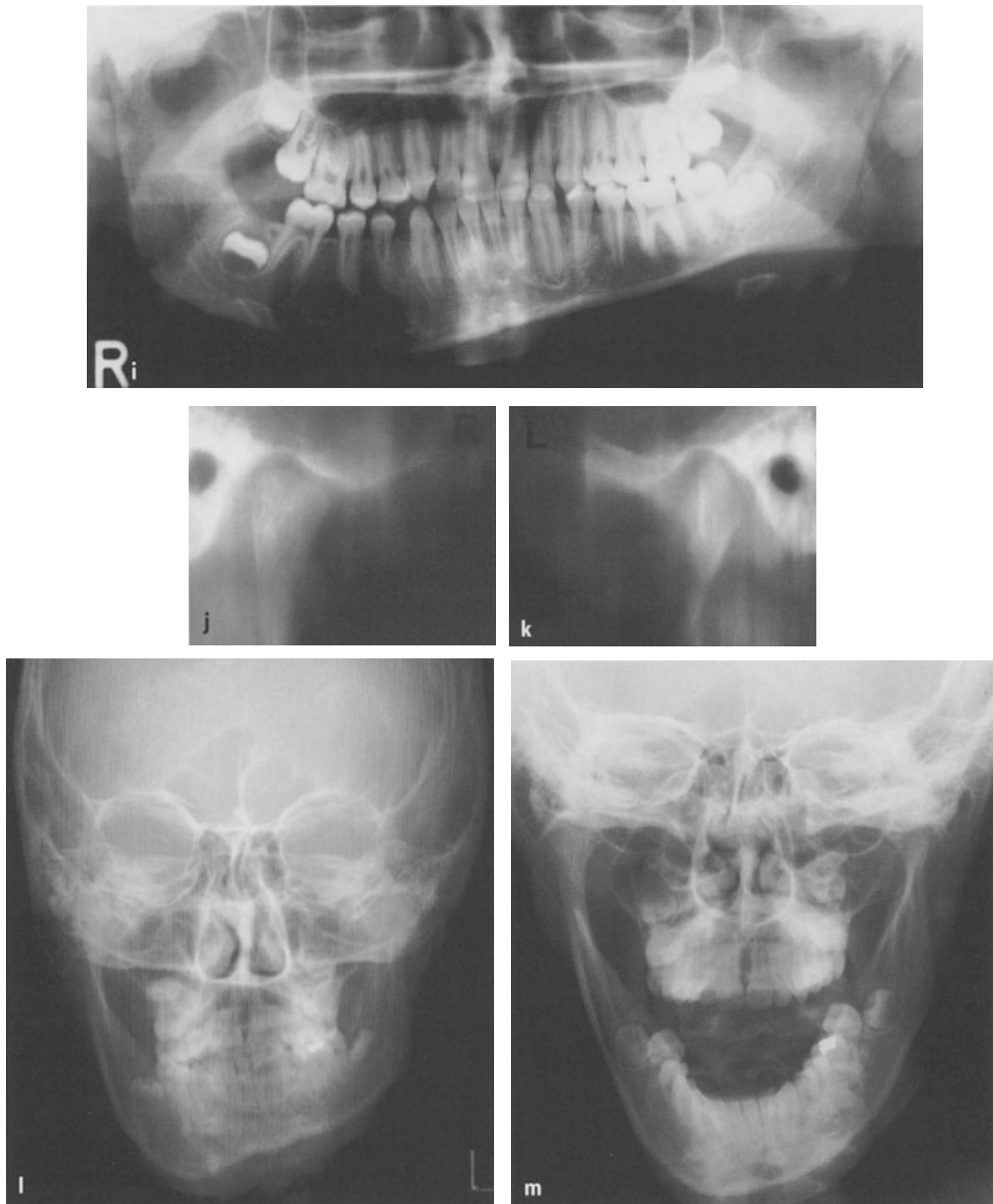


Fig. 37i-m. Unilateral juvenile H.H. and late contralateral H.E. **i** Classical panoramic view appearance of an unilateral right side H.H., at age 12. **j, k** The TMJ tomographs showing the remarkable differences in size and shape and trabeculation of the two condyles. **l** The p.a. cephalogram in the closed position discloses the severity of the facial skeletal asymmetry, with increase in height of the mandible and maxilla on the affected side and reduction in height on the other side. **m** The mandible p.a. projection at maximum mouth opening: the deformity seems much less dramatic than in the closed position. The details of the anatomical background of the deformity are more clearly visible than in the closed position. This is also true for the trabecular structure

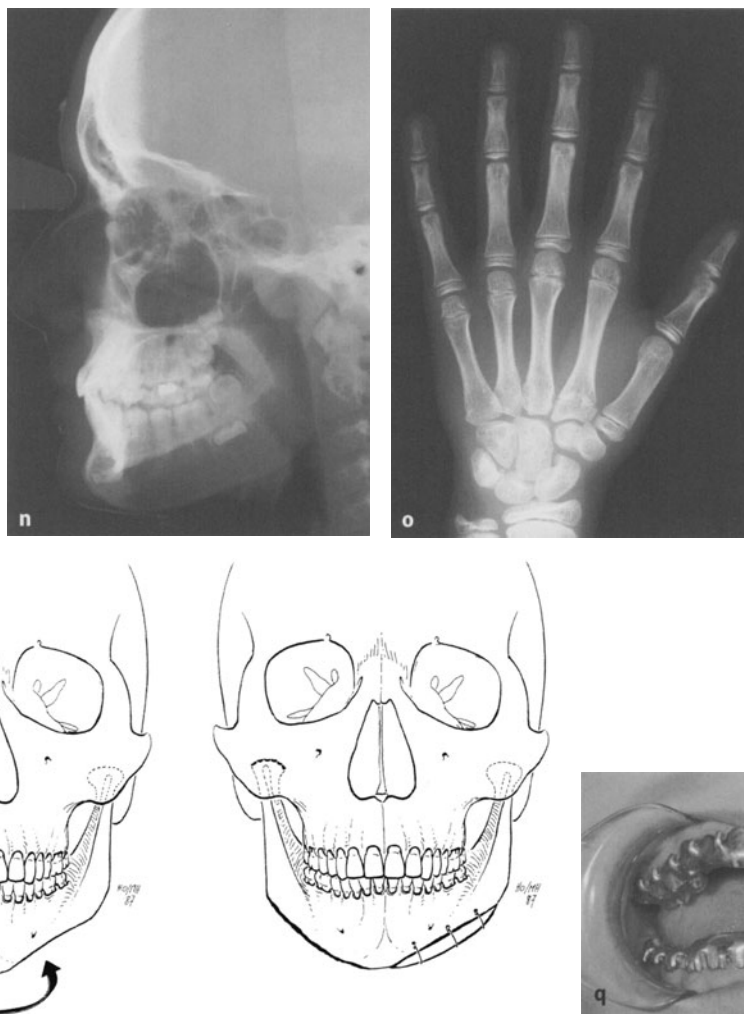


Fig. 37n–q. Unilateral juvenile H.H. and late contralateral H.E. **n** In the lateral cephalogram the discrepancy in height between the two sides is more obvious than in any other projection. **o** The hand radiograph shows the epiphyseal plate situation in accordance with the patient's age. **p** Diagrammatic illustration of corrective surgery performed: resection of the whole hyperplastic condyle and transposition of the right inferior mandible rim to the left. **q** British type of cast cap splints with training flanges are used to secure normal mouth opening

so, in these tomograms the difference in the trabecular structure between the normal and the hyperplastic side cannot be overlooked.

The *p.a. cephalogram* (Fig. 37l) shows the difference between left and right sides very impressively. In particular it reveals the difference in height of the right side of the mandible and maxilla as compared with the left and also the oblique occlusal plane.

Mandible p.a. projection in the open position (Fig. 37m): deformity of the mandible does not seem to be very impressive and yet it is very typical for this projection. There is quite an increase in ascending ramus height compared with the normal left side. The enlarged condyle, the thick neck and the coarse trabeculation are

clearly recognizable. There is deviation of the mandible towards the normal side in the open position. The “hanging down” of the maxilla on the hyperplastic side is also identifiable.

The *lateral cephalogram* (Fig. 37n) demonstrates the difference in length of the two sides of the face more precisely than does any other radiographic projection except CT. The difference is the result of the elongation of the ascending ramus of the H.H. side, whether or not the maxilla follows in such a case.

The *hand X-ray* (Fig. 37o) shows the epiphysal plates of the fingers and the radius and ulna are still wide open in accordance with the patient's age of 12 years.

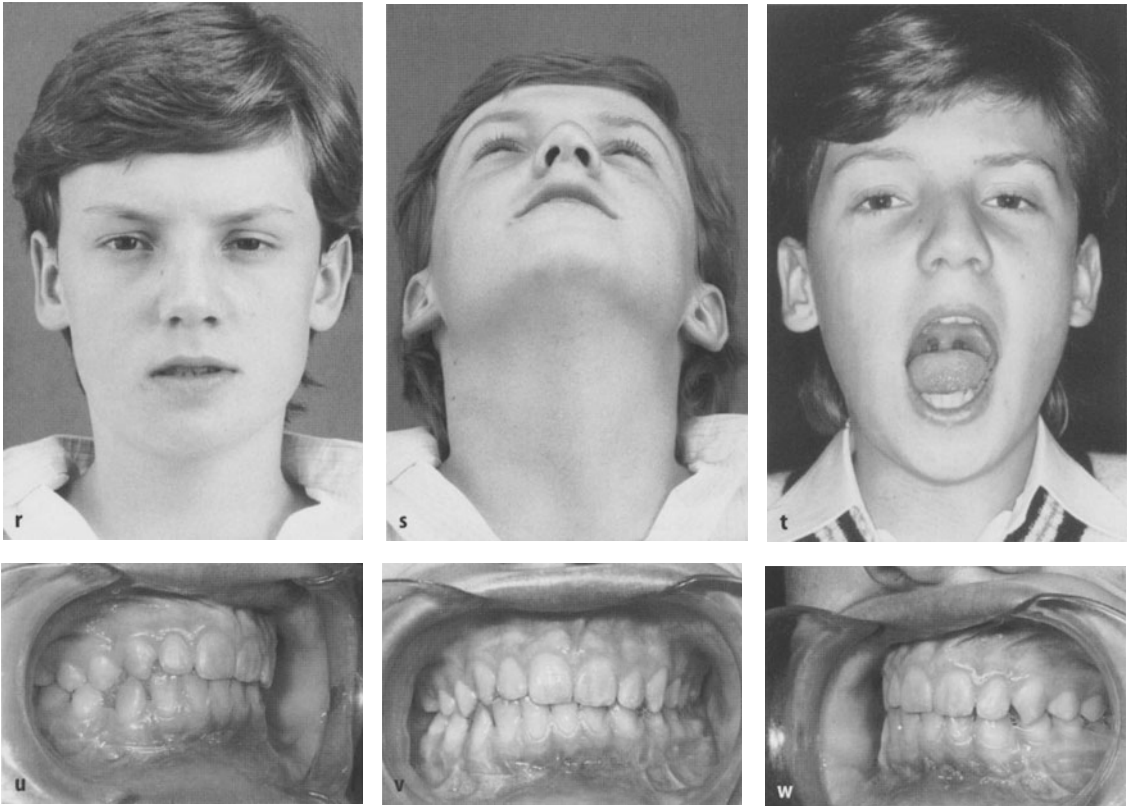


Fig. 37r-w. Unilateral juvenile H.H. and late contralateral H.E. **r-t** The patient's appearance 12 months after surgery, from in front and below, and with maximum mouth opening without deviation. **u-w** The occlusal situation 6 months after surgery, shows remarkable improvement without any orthodontic treatment

Provisional Diagnosis. Juvenile pure H.H. of the right mandible with downward growth of the maxilla and the typical rotated occlusion.

Treatment Plan. As follows:

1. High intracapsular condylectomy on the affected side and transplantation of height surplus from the right mandibular body to the lower border on the left side (Fig. 37p).
2. Postoperative insertion of a cast cap splint with training flanges for 3 months to prevent deviation on mouth opening.

Actual Treatment. The high intracapsular condylectomy was performed via a retrotragal incision. Via the oral route the convex lower border of the hyperplastic right side was resected after the mandibular nerve had been freed. This piece of resected bone was then fixed to the lower border of the left side with two wires.

Postoperative Course. There were no immediate postoperative problems. The boy was happy with his facial symmetry, he could open his mouth and had no prob-

lems with his cast cap splint (Fig. 37q). One year after surgery he had neither facial nor chin asymmetry and he had normal mouth opening without any deviation (Fig. 37r-t). Without any pre- or postoperative orthodontic treatment, the occlusion improved quite remarkably, but showed still the typical picture of unilateral H.H. (Fig. 37u-w). However, in centric occlusion his lower dental midline was slightly more to the right than before the correction. There was some reduction in the lateral translation of the mandible to the normal left side compared with that to the right side.

Four years after surgery, at the age of 16, his lower jaw had moved to the right side and his chin was also out of the midline. This had become even worse when he was checked again 8 years after surgery when the patient was 20 (Fig. 37x). The lower jaw had moved towards the side previously operated on. Now his lower dental midline was over to the right side by more than the width of a lower incisor and there was now a crossbite on the right side (Fig. 37y). The preoperatively tilted front teeth had grown completely upright. It was clear that the patient had developed a H.E. on the left side.

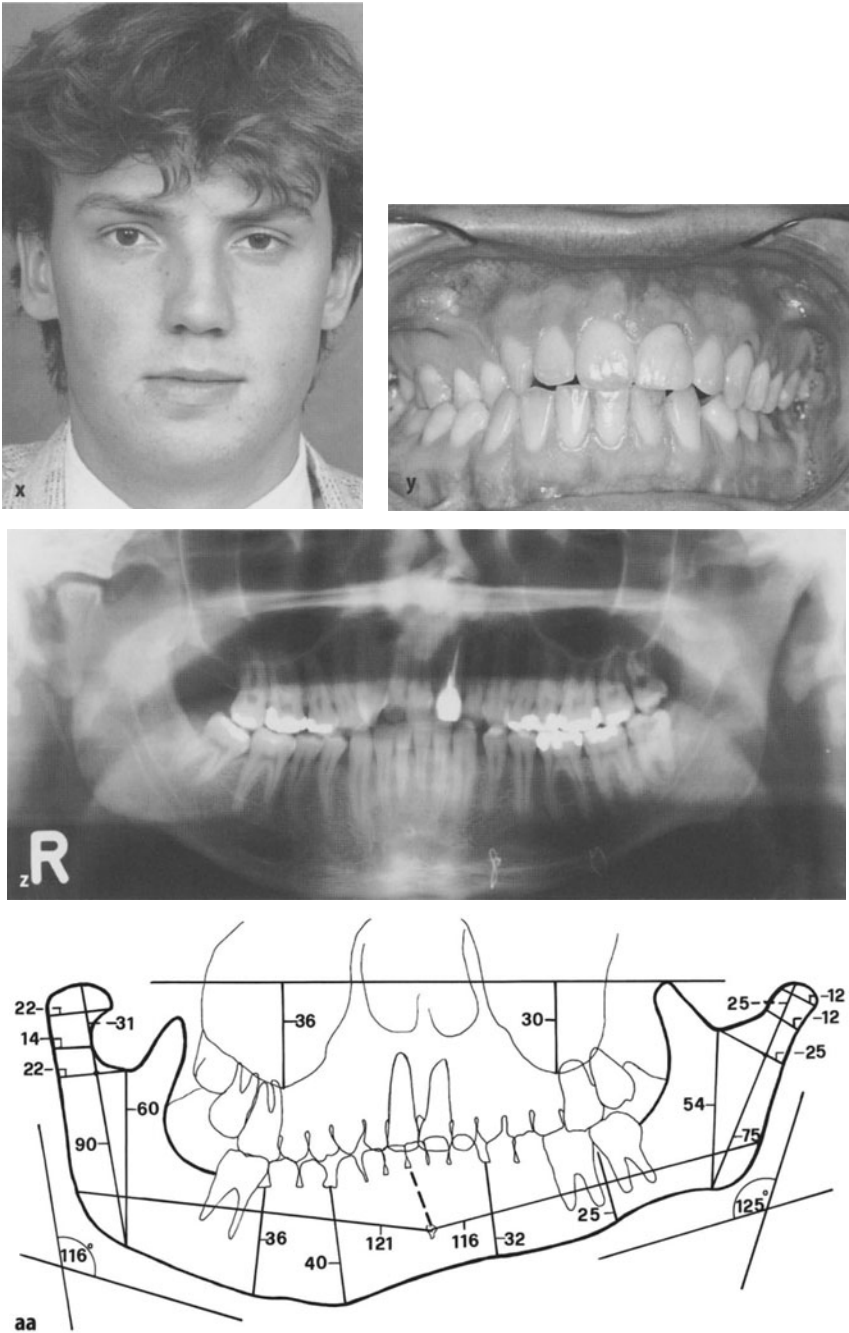


Fig. 37x-z, aa. Unilateral juvenile H.H. and late contralateral H.E. **x, y** The patient 8 years after surgery with typical appearance and occlusal picture of H.E. of the left side. **z** Panoramic view 8 years after surgery: instead of a right condyle there is a short stump of the neck with a flat sclerosed surface, and on the left side there are clear signs of a H.E., non-slender type with the lower teeth midline shifted over to the right side. **aa** Tracing and measurements of the panoramic view at age 12 years and 4 months, with typical H.H. on the right side

The OPT radiograph (Fig. 37z) now showed an almost symmetrical shape of the mandible. Its bodies were about equal in height. The lower borders showed a clear cortical covering on both sides, including the side operated on. The transpositioned right lower border had increased the left side to normal size while the hyperplastic side was reduced to a normal size. The mandibular canal on the right side was now clearly visible and the stump of the condylar neck was covered with a cortical plate which was flat on the upper surface and fitted nicely into the flat articular fossa. The left angle

was of normal configuration as was the right; the left condyle and its neck did not show any narrowing or elongation. So, the patient had developed a non-slender type of a medium degree H.E. on the left side.

The *measurements of the tracing of the preoperative panoramic radiograph* (Fig. 37aa) are a real diagnostic help in doubtful cases. In this case the correct diagnosis was possible even without measuring the tracing. There is clear elongation of the ascending ramus of the H.H. side by 16 mm compared with the normal side and 8 mm difference between the lengths of the two trunks.

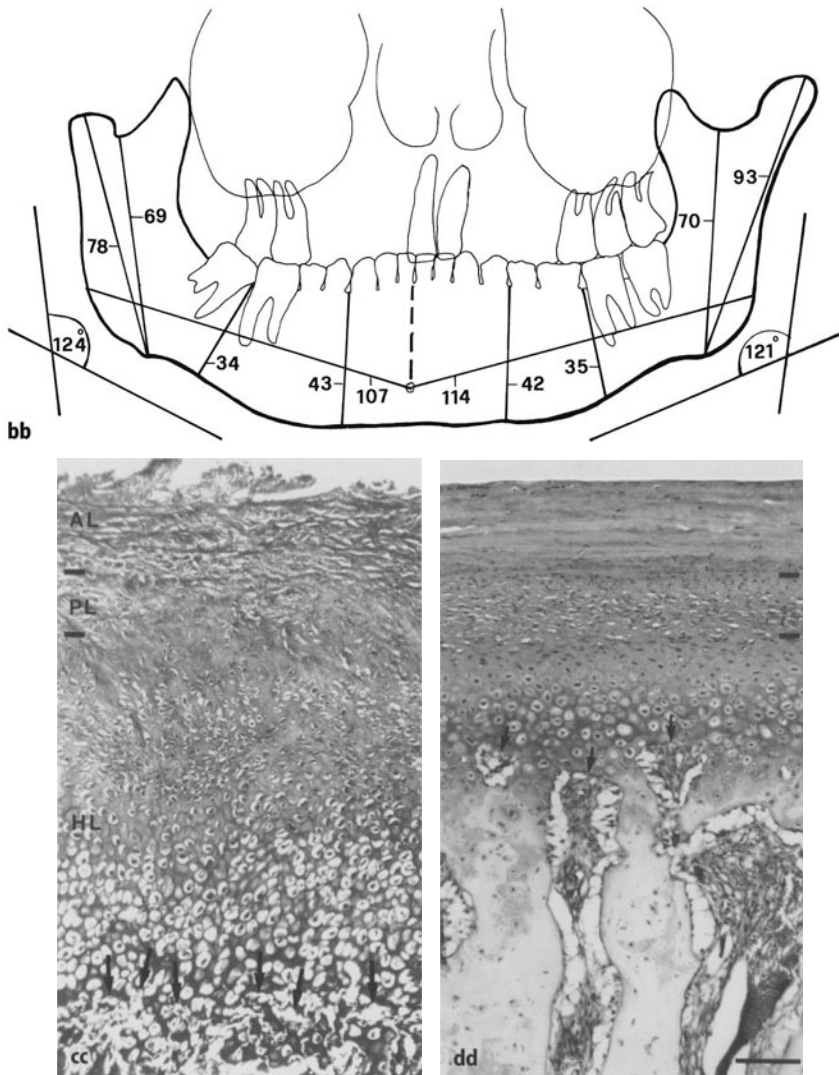


Fig. 37bb-dd. Unilateral juvenile H.H. and late contralateral H.E. **bb** Tracing and result of measurements of the panoramic view at age 20 with typical signs of left side H.E. **cc, dd** Microscopic appearance of the articular tissue of the excised condyle **cc** in comparison with that seen in a normal specimen from a female of about 10.5 years of age **dd**. Note the increased thickness of the hypertrophic layer **HL** which borders on extensive resorption lacunae (*arrows*) in the affected condyle, whereas in the control specimen only few marrow spaces (*arrows*) are in direct contact with the cartilage. (AL articular layer, PL proliferative layer; **cc** paraffin section, **dd** plastic section, toluidine blue, original magnification $\times 130$ $\text{bar} = 100 \mu\text{m}$)

A difference in length of 8 mm is recorded for the two articular processes. The difference in width of the condyles is 9 mm and the difference in height of the horizontal rami is 11 mm in the molar region and 8 mm in the premolar region indicating that there is a clear reduction in height in the molar area of the normal side, due to the pressure from the H.H. side.

The measurements of the tracing of the OPT-radio-graph 8 years after surgery (Fig. 37bb at age 20, not only permits comparison of the changes in shape of the mandible within that period, which may be partially due to the surgical correction but also partially due to spontaneous remodeling and additional new growth. The former shorter left ascending ramus is now 15 mm longer than the right. Its trunk, formerly 6 mm shorter, is now 1 mm longer. The horizontal rami are now practically equal in height, but the left is now 7 mm longer than the right. Before, it had been almost the reverse. Naturally, nothing can be said about the relation in length of the articular processes, as the right condyle had been resected. All the other length measurements tell us that the left horizontal as well as the ascending ramus has increased in length compared with 8 years earlier. There is now clear proof of the existence of a H.E. on the left side.

Histology. Compared with normal specimens, the resected condyle appeared neither enlarged nor distorted. Microscopically, the resected part of the condyle disclosed hypertrophic growth cartilage of approximately normal appearance, but increased thickness, covering the bone along the entire convex side of the specimen (Figs. 37cc). In contrast, the proliferative and articular layers along the major part of the articular surface were missing or damaged, probably as a result of surgery. Numerous lacunae at the lower limit of the cartilage indicated active endochondral ossification, although poor tissue preservation prevented identification of chondroclasts and osteoblasts (Fig. 37cc). Trabeculae of the subchondral spongiosa containing cartilage islands down to the resection border were thin, lacked well-defined orientation, and formed a tight network around unusually small marrow spaces (Fig. 79b).

Diagnosis. Actively growing condyle with dysplastic spongiosa characterized by a tight network of haphaz-

ardly arranged trabeculae containing extensive cartilage islands.

Comment: The structure of the subchondral spongiosa seen in this case appeared characteristic of H.H. Cartilage remnants down to the resection border at a distance of almost 6 mm from the zone of erosion suggest markedly increased endochondral ossification. Taken together, the observations are indicative of a condylar hyperplasia at the stage of on-going excessive enlargement.

Discussion. This case presents, at the age of 12 years, as a fully developed unilateral pure H.H. which must have started very early in infancy or childhood. Whether it already existed at birth is difficult to prove, although the patient's father, a medical practitioner, had noticed an asymmetry of the chin and then an occlusal abnormality during the deciduous dentition period. The very flat surface of the condyle and also of the glenoid fossa does suggest that during the development of these anatomical structures an abnormal influence on the formation of the glenoid fossa was present.

After high condylectomy at the age at 12 years and 4 months and the transfer of the right lower border to the left side, normal further mandibular growth and development proceeded for about 3 years. The left side then became hyperactive and produced a unilateral H.E. of the non-slender type. The fact that the downward-grown maxilla was not surgically corrected had no negative influence of any kind.

Histologically, the specimen of the resected part of the condyle seems to show the typical picture of the existence of condylar hyperactivity of the H.H.-type.

Conclusion. This case raises the question whether a unilateral H.H. can start to develop before birth, as the father had noticed a chin asymmetry present at birth. It must have definitely existed in infancy as the father had diagnosed an occlusal abnormality at the time of the deciduous dentition. This case also shows that the contralateral side can later, and obviously independently, develop condylar hyperactivity due to stimulation by either the M or the L condylar growth factor, or it may even be a hybrid form. Indeed, this abnormal condylar growth regulation is a very interesting growth physiology subject.

12.2.4

Differential Diagnosis of Unilateral Hemimandibular Hyperplasia

Unilateral H.H. which affects one side of the mandible and terminates at the symphysis, must be clearly distinguished from condylar enlargement of any other origin (A. Papavasiliou et al. 1983) which can present a similar clinical picture.

There exist several other aetiological possibilities which can produce an enlargement of the condyle of abnormal size and shape. It may be a simple osteogenic enlargement of the condyle as described in case Fig. 39. Enlargement and deformation due to a tumorous lesion or of arthrotic origin must also be considered (see case Fig. 38). They all cause an increase in volume of the condyle, contrary to those aetiological factors which result in a damage to the condyle. Different again is the recent-

ly described unilateral dysplasia of the mandibular condyle (H. Tank et al. 1998).

As the ramus-condyle length increases through the tumorous enlargement of the condyle and thereby pushing the mandibular body with its teeth further inferiorly, producing an open bite, the inferior alveolar height hardly increases. The upper teeth may follow downwards, preventing a severe open bite on that side, but this is also rather rare. Pseudo-H.H. cases may show, on the radiographs, an enlarged condyle but no height increase in the ramus or body. None of these aetiologies of a condylar enlargement produces a downward convexity of the inferior border of the mandible. This is the main differential diagnostic sign.

If soft tissue volume produces a similar clinical picture, then the panoramic radiograph will readily disclose the truth. None of the pseudo-cases can show symptomatology of M-growth regulator hyperactivity.

Simple Osteogenic Hyperplasia of the Condyle

This pathology involves only the condyle. It becomes homogeneously enlarged, showing radiographically a uniform fine trabeculation with some localized sclerosis. The shape of the condyle may become slightly abnormal but not in the way the condyle of a H.H. case does. The condylar neck does not become longer and thicker.

Clinically the facial appearance looks very similar to a H.H. abnormality. There is a marked increase in the height of the affected side of the face. The prominence of the chin may be shifted over to the normal side; this is not so in a pure H.H. case. The inferior border of the affected side of the mandible is normal and not bowed inferiorly and the histology of such a hyperplastic condyle shows an almost uniform picture of fine trabeculation. There are no signs of former hyperactivity of the cartilaginous zone.

Unilateral Pseudo H.H. Due to Enlargement of the Condyle Caused by Deformative Arthrosis (Fig. 38)

Sex and Age. Female, 23 years of age.

Aetiology and History. The patient had never been really ill. About 3 or 4 years previously she had had TMJ-clicking on the right side and simultaneously she had noticed that her face had become asymmetrical. She had no idea why this had occurred.

Clinical Findings. Looking at the patient's face from in front (Fig. 38a), there is a very remarkable difference in the height of the two sides of the face, the right side being much higher than the left, and flat. Its mandibular lower border seems downwardly convex and rotated medially. The chin is slightly out of the midline to the left. The left side has a normal configuration of the

Arthrotic enlargement of the condyle with exostosis is the most common cause of such an enlargement without any growth influence. It can produce a similar clinical picture to the exclusive true hyperplasia of the condyle. However, radiologically and histologically it differs quite distinctly. It is sclerosed with exostoses and islands of dense opacity, resembling eburation.

Benign tumours of the condyle may also produce a similar clinical appearance such as fibro-osteoma, fibroma, myxofibroma, chondroma, eosinophilic granuloma or giant cell tumour, etc. These tumorous lesions may be localized to the condyle only or also affect the neck and ramus.

All these tumour-like lesions cannot be confused with H.H. as the radiological and clinical pictures differ remarkably. They produce no increase in mandibular body height with a characteristic trabecular structure and they show no downward bowing of the inferior border. They also do not produce the often typical kink at the symphysis. Tumorous neoplasms within the horizontal ramus like fibrous dysplasia, endosteal angiomata, von Recklinghausen's disease or hemihypertrophy of the mandible (D. Jacquemaire and J. Delaire 1983) can also push the inferior mandibular border downwards, thus imitating a unilateral H.H. However, the radiographs will immediately disclose the nature of the deformity.

Unilateral masseter hypertrophy might simulate an appearance similar to a unilateral H.H. Again several classical signs of the product of hyperactivity of the growth regulator M will be missing. The condyle and its neck are not enlarged. The horizontal ramus lacks the inferior bowing and there will be no increase in its vertical height and no tilting of the teeth. But a rounded angle, similar to that of a H.H. may be present.

cheek. Its mandibular lower border seems shifted a little laterally and rotated outwards. The mouth opening is normal, but with clear deviation to the left side. Soft tissues and facial nerves were normal.

The occlusion (Fig. 38b) shows an open bite on the right side, beginning at the midline. There seems to be a slight tilting of the lower front teeth towards the side of the open bite. The midline of the lower teeth was pushed 3 mm over to the left side. She had no TMJ problems.

Radiographs. The panoramic view did not exist when the patient came for treatment. Lateral view projection of both mandibles showed, apart from enlargement of

the right condyle, a normal configuration on both sides. The TMJ tomogram including the ascending ramus (Fig. 38c) shows a normal ramus. The condylar neck is rather slim but of normal length. The condyle itself was enlarged and has an irregular surface and trabecular structure. Sclerosed areas and irregular translucent ones are visible.

Provisional Diagnosis. Enlargement of the right condyle of uncertain origin producing remarkable facial asymmetry in height, and an open bite on the same side.

Treatment Plan. Resection of the whole condyle.

Actual Treatment. A retrotragal incision approach was used to give the necessary access to the TMJ area for the resection of the whole condyle. A normal occlusion immediately resulted (Fig. 38d). This specimen was not at all like a normal condyle. It had a long anterior “beak” and a very irregular surface (Fig. 38e).

Thirty six years after surgery the patient was kind enough to come for a follow up. A panoramic radiograph (Fig. 38f) was taken which reveals both sides of the mandible to be equal except for the missing right condyle and neck.

The measurements of the tracing (Fig. 38g) of that panoramic radiograph reveal absolutely equal mandibles. Both horizontal rami as well as the trunks are equal in length and so is the height of the two horizontal rami. The only difference is the missing articular process on the right side.

Histology. The histological diagnosis of the Institute of Histopathology (Prof. Rüttner) of the University Hospital, Zürich, was: deformative arthrosis of the resected condyle.

Discussion. A tumour-like enlargement of the condyle can develop due to several aetiologies, one is arthrotic influence. It produces vertical increase in the ascending ramus area on the same side, simulating a true unilateral H.H. However, the very irregular surface of the condyle, visible on the radiograph, and the very irregular structure, with clearly enclosed areas of sclerosis as well as translucency, indicated that this was not a H.H.-appearing condyle. Also, the shape of the whole mandible meant that it could not be a H.H., although in such a case a positive finding of cellular hyperactivity in the scintigram would have had to be expected due to the inflammatory arthrotic reaction in the condyle. The panoramic view taken 36 years after surgery proved that

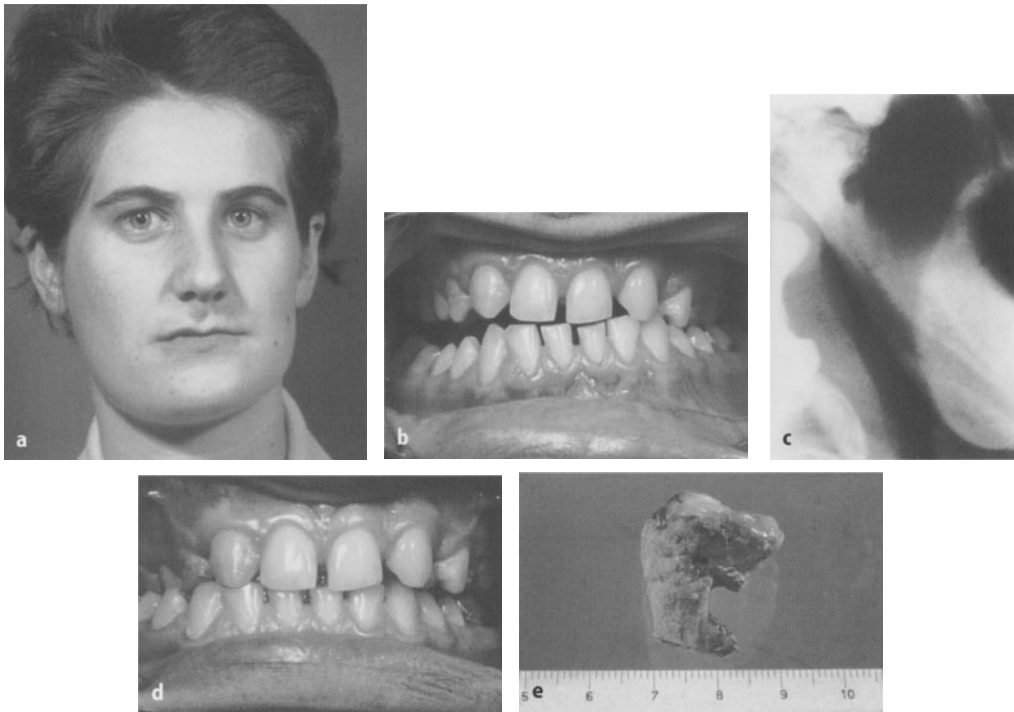


Fig. 38a–e. Unilateral pseudo H.H. due to enlargement of the condyle because of deformative arthrosis. **a** The patient's appearance is just like that of an unilateral H.H.: one side is remarkably longer than the other, it is flat and the inferior border seems more medial than that on the other side. **b** The occlusal picture shows an open bite on the longer side of the face and tilting of the teeth towards it. **c** The tomogram of the ramus shows a clear enlargement of the condyle only. It has an irregular surface and structure with an anterior beak. **d** After resection of the condyle normal occlusion resulted. **e** The resected condyle confirms the radiological picture

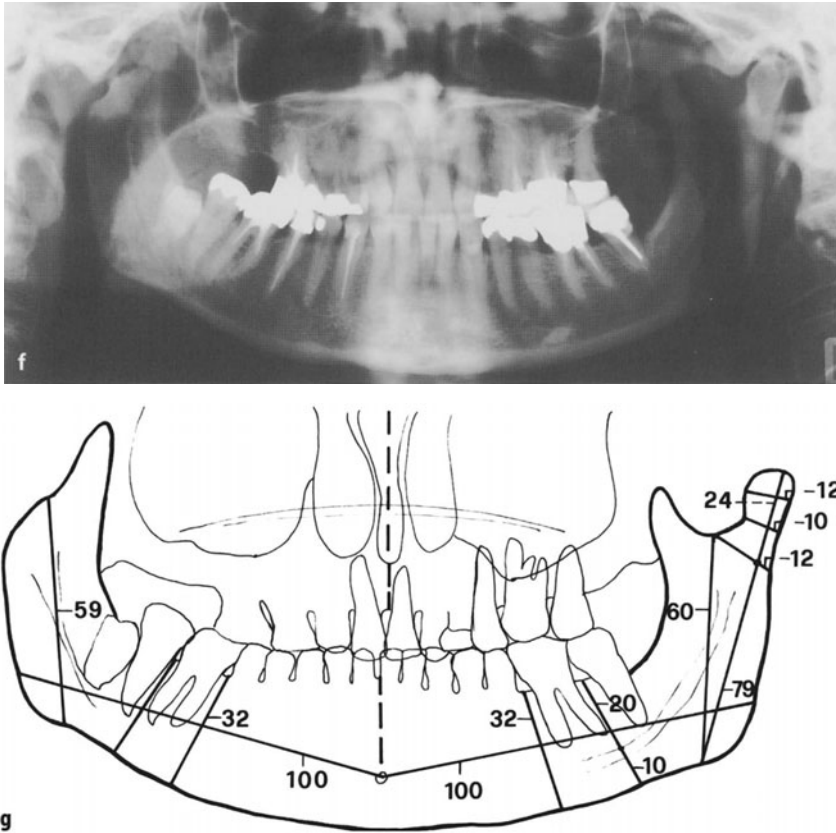


Fig. 38f, g. Unilateral pseudo H.H. due to enlargement of the condyle because of deformative arthrosis. **f** The panoramic view taken 36 years after condylectomy, discloses an absolutely normal mandible in which the condyle and its neck is missing. There is normal occlusion. **g** The measurements of the tracing of Fig. 38f show the two absolutely equal halves, except for the missing right articular process

there were no signs of a former H.H. anomaly. Both halves of the mandible were absolutely equal.

Conclusion. An enlargement of the condyle due to deformative arthrosis or any bony tumour can easily simu-

late the outward appearance of an unilateral H.H. But that can always be excluded as long as there is no increase in height in the molar-bicuspid area of the mandible on the affected side and no downward convexity of its lower border.

Unilateral Pseudo H.H. due to Simple Hyperplasia of the Condyle (Fig. 39)

Sex and Age. Female, 31 years of age.

Aetiology and History. Patient had no idea why and when the facial deformity started and when it ceased. The patient had no pain in the TMJ region or the facial musculature. She was referred by her dentist because of an open bite on the right side.

Clinical Findings. There is a facial asymmetry with the right side longer and flatter than the left (Fig. 39a). The right angle area is less prominent and lower than on the left. This is also true for the right lower border of the

mandible. The chin is shifted towards the left side (Fig. 39b). Facial nerves and musculature are normal. The lip commissure is slightly oblique with the right corner a little lower and more medially than the left. The mouth opening is within the normal range but with deviation of the mandible to the left side. There is no real occlusion because of a rather severe open bite on the whole right side and a crossbite on the left (Fig. 39c). The midline of the lower teeth is also slightly over to the left side.

Radiographs. The panoramic view discloses the skeletal background of the deformity (Fig. 39d): The mandible has two different ascending rami, angles and condyle forms. The right condyle is slightly enlarged and its neck has a thickened appearance. It has a slight partially sclerotic and partially translucent structure. The right ascending ramus is steep and elongated compared with the left and forms, together with the body, an almost rectangular angle. The right and left horizontal rami are of almost equal height with an equally thick cortical covering. There is no increase in cancellous or alveolar bone. There is no inferior convexity of the right mandibular lower border, rather the contrary. The mandibular nerve runs at a normal distance from the dental apices on both sides. The left angle area is rather flat. The left ramus and condyle seem normal. The base of the maxilla is horizontal and there is no downward growth of the teeth on either side.

Mandible p.a. projection in maximum opening. In spite of its very poor quality it was still possible to visualize the following: the very pronounced elongation of the right ascending ramus including the condyle and neck was very obvious and also the shift of the mandible to the left side. The left ramus and angle region were rotated outwards which brought the anterior crest of the ramus with the coronoid process more medially. The right ramus seemed to have done the reverse. The chin prominence was far out of the facial midline to the left. The maxilla was slightly hypoplastic on the right. Also the nasal and paranasal sinuses seemed to be different in size, the right ones smaller than the left.

The TMJ tomograms including the rami (Fig. 39e,f) show the described difference between left and right very clearly: the left condyle and ramus appear normal, but the right condyle and neck are uniformly enlarged and show the above mentioned sclerotic structure as far down as the base of its neck.

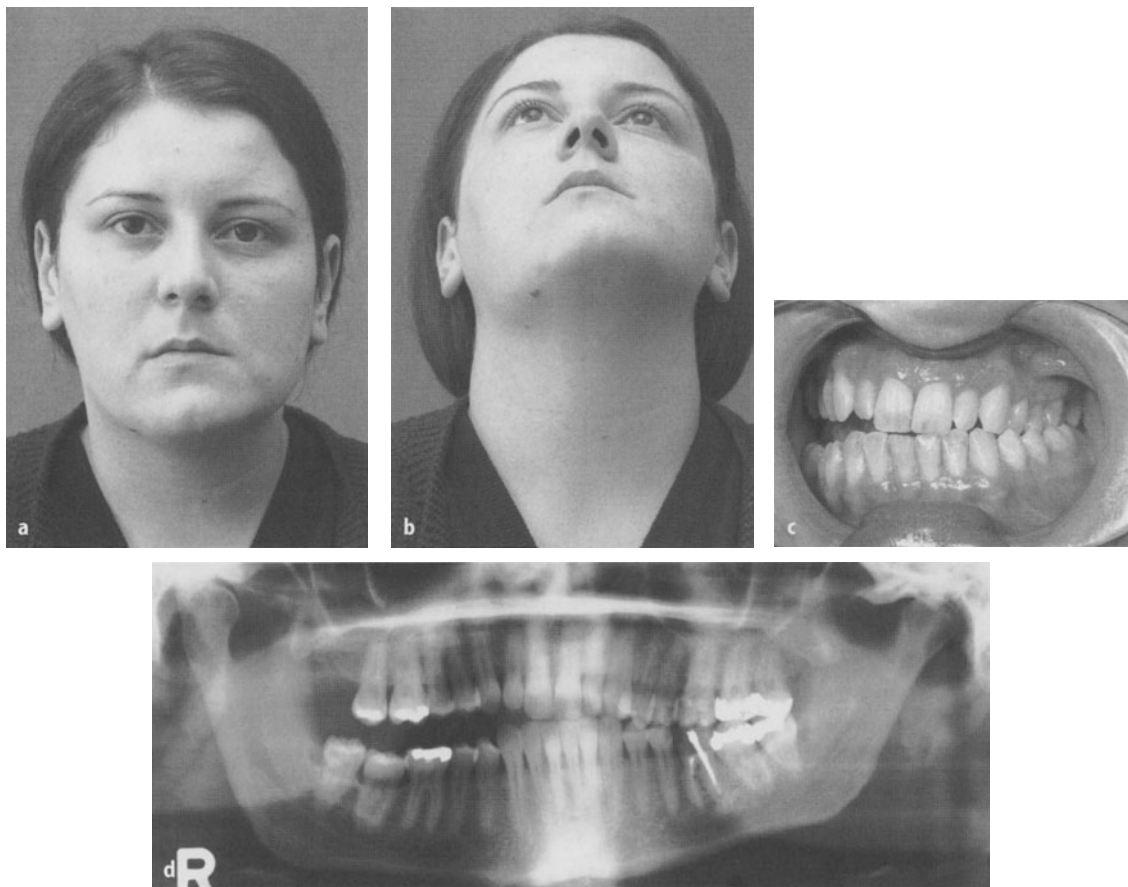


Fig. 39a–d. Unilateral pseudo H.H. due to simple hyperplasia of the condyle. The patient's **a** front appearance and **b** from below looks very similar to a H.H. deformity. **c** Severe open bite on the elongated right side and lateral crossbite on the left side. Slight tilting of the lower front teeth towards the elongated side. **d** The panoramic view discloses that this cannot be a H.H. mandible, in spite of the enlargement of its condylar head and neck: no downward bowing of the inferior rim, no height increase in the horizontal ramus, no large rounding of the angle

The lateral cephalogram (Fig. 39g) shows the open bite and the steep right ascending ramus and also that the right body extends further down than the left. There is no convexity of the lower border on either side.

The measurements of the tracing of the OPT radiograph (Fig. 39h) demonstrate the following differences between right and left mandibles: the right ascending ramus is 13 mm longer than the left, with a difference of 5 mm between the trunks and 7 mm between the length of the articular processes. The right horizontal ramus is 11 mm longer than the left. The height of the horizontal rami is equal on both sides and so is the inferior extension of the maxillary sinuses. The two angles differ by 13° .

Provisional Diagnosis. Simple hyperplasia of the right condyle, of unknown origin.

Treatment Plan. According to the model operation it will not be sufficient to resect the enlarged condyle

alone. A sagittal splitting procedure on the ramus on the contralateral side will be necessary additionally to achieve a good occlusion.

Actual Treatment. The operation was carried out as planned. It was found that the resected condyle had a very irregular surface with large irregular spots and streaks of whitish hard material (Fig. 39i).

Histology. Macroscopically, the removed condyle was enlarged, its antero-posterior diameter being 13 mm. In the posterior-superior region, it appeared deformed due to a protuberance projecting in the posterior-superior direction similar to an osteophyte. Microscopically, the articular surface of the specimen revealed fibrocartilage which, however, lacked a distinct superficial, intermediate, and deep zone along most of its extent. In these areas, the articular surface exhibited, in part, damage presumed to have resulted from surgery and, in part, fibrillation indicative of beginning osteoar-

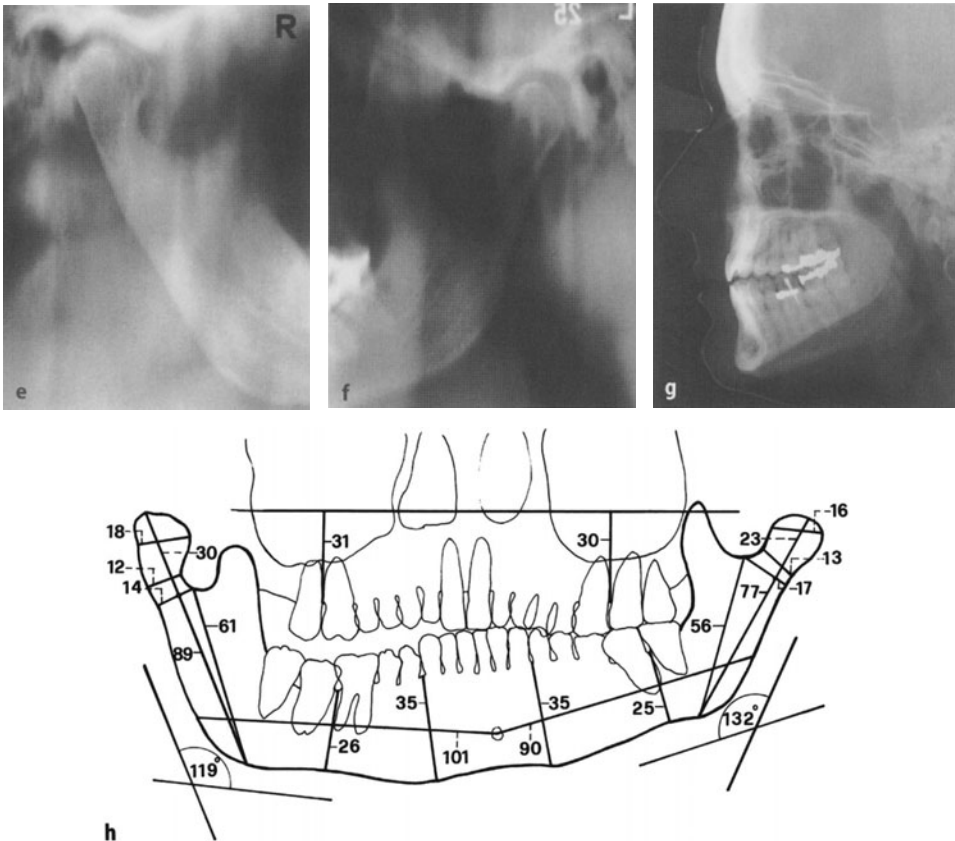


Fig. 39e–h. Unilateral pseudo H.H. due to simple hyperplasia of the condyle. **e, f** The TMJ ramus tomograms permit clearly the visualization of the differences between the two condyles, in both shape and structure. The enlarged right condyle and neck could, radiologically, be a hybrid-hyperplasia type. **g** Even on the lateral cephalogram, the diagnosis of H.H. can be excluded in spite of the existing difference in height between the two horizontal rami: the downward bowing of the inferior rim is missing! **h** The tracing of the OPT and its measurements disclose interesting facts

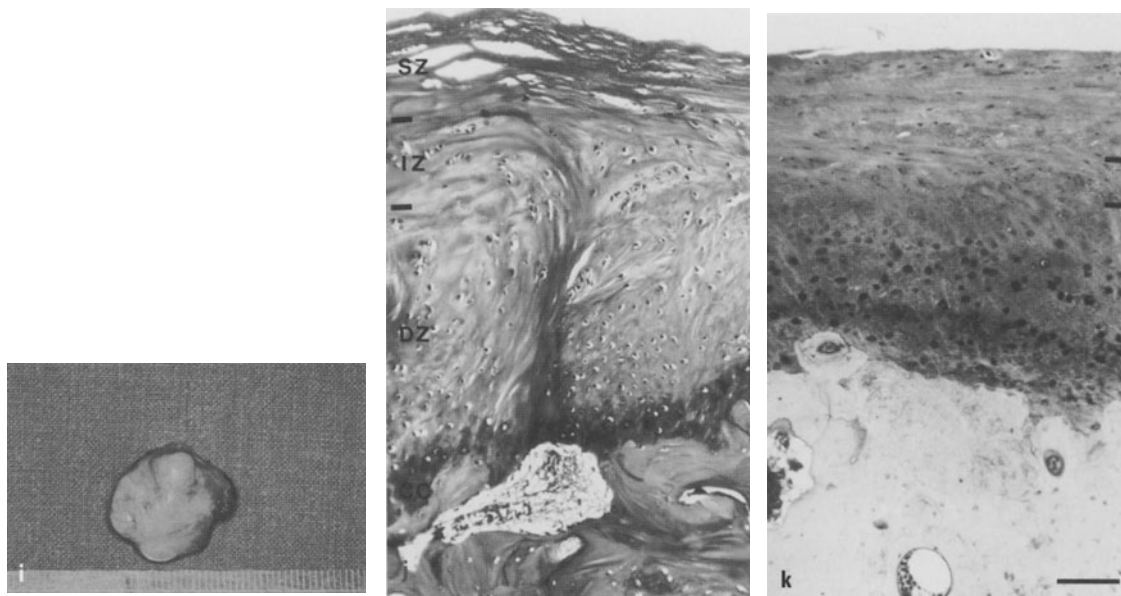


Fig. 39i-k. Unilateral pseudo H.H. due to simple hyperplasia of the condyle. **i** The resected condyle has an irregular surface with hard whitish prominences. Microscopic appearance of the articular tissue of the excised condyle **j** in comparison with that seen in a normal specimen from a female of 31.5 years of age **k**. Although the articular tissue of the affected condyle **j** is slightly thicker and consists almost entirely of fibrocartilage, it does not differ significantly from the tissue seen in the normal specimen **k**. (SZ superficial zone, IZ intermediate zone, DZ deep zone, CC calcified cartilage; **j** paraffin section, **k** plastic section, toluidine blue, original magnification $\times 130$, bar = 100 μm)

throsis (see Fig. 79k). Subjacent to the surface fibrillations, the density of chondrocytes was low. Frequently the tidemark (uncalcified-calcified cartilage junction) as well as the osteochondral junction followed an irregular, scalloped course (Fig. 39j), and in one place, the tidemark was duplicated, suggesting active progressive cartilage remodeling. Although some small marrow spaces were located directly at the osteochondral junction (see Fig. 79k), the subchondral bone plate as well as the density and architecture of trabeculae in the spongiosa appeared inconspicuous. In the region of the protuberance, the fibro-cartilage was thicker than along the rest of the articular surface and disclosed the typical layered appearance. In this area, the subchondral bone appeared sclerotic, virtually lacking any trabeculae, and also contained cartilage islands which, however, were confined to a relatively small tissue band subjacent to the osteochondral junction.

Diagnosis. Grossly enlarged condyle exhibiting initial, mild osteoarthrotic changes.

Comment: The observations in this case are similar to those in adult cases of H.H. In particular, no indication of a tumour, e.g. an osteoma or osteochondroma, could be detected. Thus, there seems to be no histological feature allowing distinction between simple condylar hyperplasia and H.H. in adult patients.

Discussion. A tumour-like bony enlargement of the condyle and its neck, named by M. Rushton (1944) "simple hyperplasia of the condyle", can exist without causing any abnormal growth of the same side of the mandible. Although it produces almost the same outward deformity appearance as does unilateral H.H., it does not cause any additional bone production either in the ascending or horizontal ramus. It also does not produce the downward convexity of the lower border of the mandible nor the classical large rounding of the angle, both very typical of H.H. On the contrary, the angle becomes almost rectangular. There is no increase in the body height and no downward displacement of the mandibular canal. These facts seem to prove my hypothesis that H.H. produces bone mass and that the increase in the height of the horizontal ramus is not due only to a compensatory upward growth of the alveolus.

In this case, the measurements of the tracing of the panoramic radiograph (Fig. 39h) prove very clearly that the right ascending ramus, independent of the enlarged condyle, is longer than the left. Also the right horizontal ramus is longer than the one on the normal side. I have no other explanation for this than that the enlargement of the condyle happened in addition to an already existing H.E. on that side. This is, of course, only guesswork as I have no proof for it other than the existence of the severe deviation of the chin to the other side. The tumorous enlargement of the condyle alone should not be

able to cause such a degree of deviation. In the case of Fig. 38 there is a tumorous enlargement of the condyle of another origin to about the same size, also producing an open bite of the same amount, but with equal lengths of the ascending and horizontal rami. That case permits the assumption that it possibly has H.E. as the origin of the elongation of the horizontal as well as the ascending rami, on the side of the tumorous enlargement of the condyle, and was not due to that pathology.

The histology of the resected part of the very enlarged condyle including its neck disclosed no specific alterations of normal fine trabeculation, except a circumscribed superficial osteoarthritis. The same picture is found in condyles of H.H. cases when abnormal growth has come to a stop. This histological finding leads to the conclusion that the condyle and its neck can become remarkably enlarged without any stimulating influence on the two growth regulators and yet produce a radiological picture of the condyle similar to a H.H. or a hybrid growth anomaly. For these reasons we have to include such an anomaly of the condyle in the category of "simple hyperplasia of the condyle".

Neurofibromatosis Producing an Appearance Similar to Unilateral H.H. (Fig. 40)

Sex and Age. Female, 31 years old.

Aetiology and History. Facial asymmetry had already been noticed in infancy. There was also a surplus of flabby skin, enlargement of the right ear and lobulation of the right side of the tongue. Within some years the increase in facial height on the right side became very disturbing. Several operations had been performed for removal of surplus tissue. The patient then came for detailed information regarding the prognosis and possible improvement.

Clinical Findings. There was severe facial asymmetry (Fig. 40a) due to vertical elongation of the right side of the face with the chin prominence out of the midline, to the same side, by almost 20 mm. The elongated right side seemed slightly more voluminous than the rather flat left side, which tended to be in a straight oblique line to the deviated chin prominence. On palpation the right surplus in length was rather soft. The "chin prominence" could easily be pushed away, to either side or even "removed" by pulling the skin of the cheek cranially.

There were several tumour-like nodes on the right side of the very irregularly formed tongue.

Mouth opening was unimpaired. The upper and lower jaw were edentulous, except for one carious right lower molar.

There were several café-au-lait spots on the skin on various parts of the patient's body.

Conclusion. Any enlargement of one condyle of any origin may produce almost the same outward deformity appearance as does an unilateral H.H. However, in order to diagnose a true H.H. it is necessary to have additional bone production which makes the horizontal ramus on the affected side higher and its lower border convex downwards. In a non-growth-stimulated enlargement of the condyle there is no rounding of the angle and the downward displacement of the mandibular nerve. The latter, however, can be absent in cases of a very mild form of hyperactivity of the M-growth regulator, or when it has developed over many years, as seen in Fig. 32.

The elongation of the ascending ramus in this case, independent of the enlarged condyle, and also the elongation of the horizontal ramus compared with the contralateral normal side, both clearly found on the tracing of the OPT, cannot be explained by the tumorous enlargement of the condyle alone. It might be that the tumorous growth started on the basis of an already existing H.E. anomaly.

Radiographs. The panoramic view is very striking: there is not the slightest chance of diagnosing it as a unilateral H.H. There is very little of the ramus and body left on the right side of the mandible: no increase in height of the horizontal ramus and no increase in ramus length, nor enlargement of the condyle and neck. There are just rudimentary parts of that side of the mandible recognizable.

Provisional Diagnosis. Appearance simulating the picture of unilateral H.H., caused by severe destruction of the right mandible, including the ascending ramus affected by von Recklinghausen's neurofibromatosis.

Treatment Plan. Surgical correction of the deformity by massive soft tissue excision and partial resection of the affected side of the mandible, with simultaneous reconstruction using an iliac crest graft.

Actual Treatment. The patient refused any treatment. She had come from another European country.

Discussion and Conclusion. There are several possibilities for pathology to simulate the appearance of an unilateral H.H., as mentioned earlier. However, the radiograph presents no chance of misdiagnosing such a case as a unilateral H.H. But its corrective surgery could be

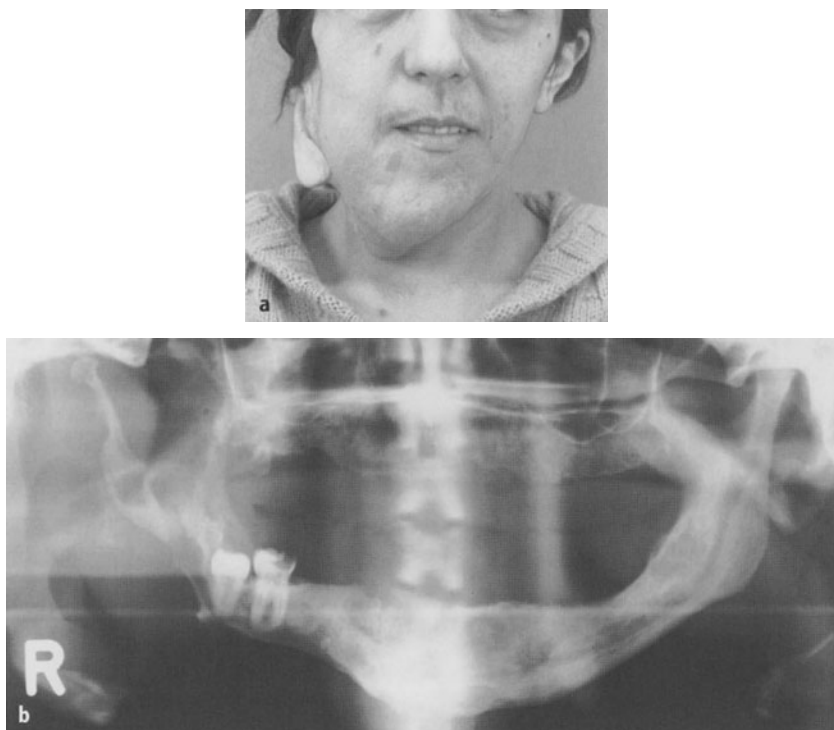


Fig. 40a, b. Neurofibromatosis producing an appearance similar to an unilateral H.H. **a** Elongation of right side of the face similar to unilateral H.H. **b** The panoramic radiograph discloses a pathology of the mandible which excludes automatically any possibility of a H.H.

quite hazardous, with not only the possibility of causing damage to the branches of the facial nerve but also of running into severe bleeding.

In a case like this there could also be a vascular abnormality around the mandible as found in the two patients of Fig. 20. Also, a massive fibro-angioma can pro-

duce a similar picture, not only a perimandibular cavernous haemangioma. Before any surgery is performed, even before a biopsy is taken, one has to be sure that there is no extensive abnormal vascularization involved. Aspiration in various parts of the tumorous mass or, even better, angiography can make things clear.

Unilateral Pseudo H.H. due to Unilateral Masseter Hypertrophy (Fig. 41)

Sex and Age. Male, 22 years of age.

Aetiology and History. Patient could not remember when the asymmetry started. Recently, his friends had told him that he had an asymmetrical face.

Clinical Findings. From in front, he presents a marked difference in configuration of the right and left halves of his face (Fig. 41a). The right side seems unobtrusive. The left side is very bulky in the angle area. The bulk is not only more voluminous on the lateral aspect but also the lower border of the angle area extends further downwards. The swelling is rather hard but not of bony consistency above the angle. The angle itself protrudes slightly laterally and is of bony consistency. There is a normal oc-

clusion. When closing the teeth forcibly the swelling in the angle area becomes more voluminous. The facial nerves were normal and so was mouth opening.

Radiographs. The panoramic view (Fig. 41b) shows a rather strong mandible with a configuration similar to a possible bilateral H.H. The whole mandible looks very voluminous in all sections of the horizontal as well as the ascending rami. This also includes the condyles and their necks and even the two mandibular canals. The right angle is roundish but of a small diameter and yet almost rectangular.

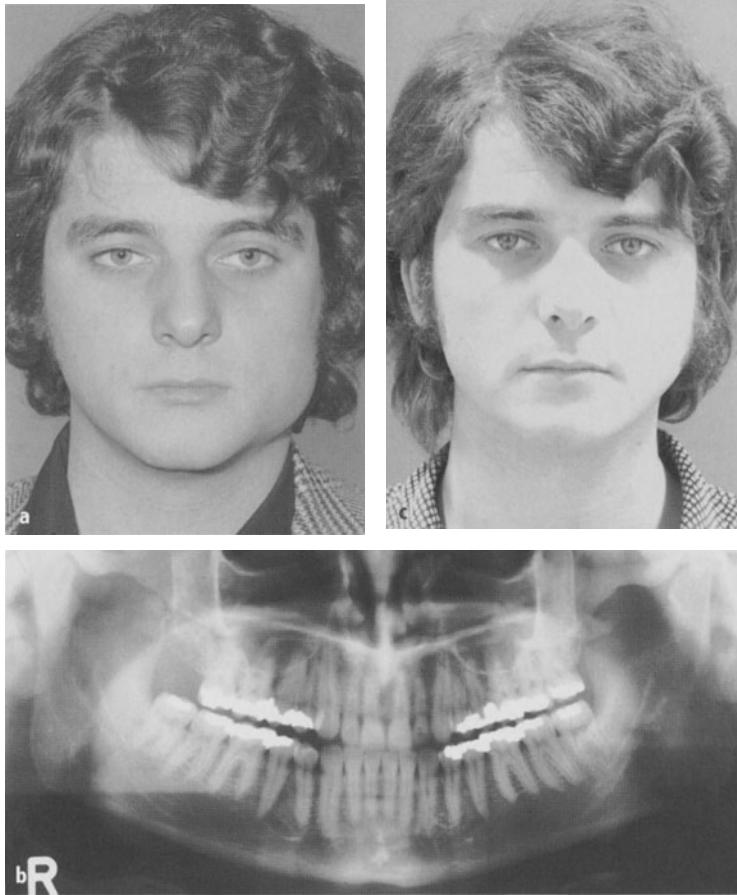


Fig. 41a–c. Unilateral pseudo H.H. due to unilateral masseter hypertrophy. **a** The patient's front view discloses a marked swelling in the left angle area. **b** On the panoramic radiograph the mandible seems very strong but symmetrical, except for the very prominent and deformed left angle. Both coronoid processes look very short and obtuse. **c** The patient's appearance 18 months after surgery

The left angle protrudes more laterally and downwards than the contralateral side, thereby producing an antegonial concavity. The structure of both sides is within normal limits. The lower border on either side is almost horizontal, without any downward convexity. The mandibular canal is not displaced downwards to the lower border. There is an irregular translucency in front of the very enlarged and deformed left angle.

The *p.a. cephalogram* shows an almost symmetrical facial skeleton except in the left angle area of the mandible. There is a laterally protruding bony contour with a cyst-like irregular translucency close to the lateral cortical plate.

Provisional Diagnosis. Unilateral masseter hypertrophy on the left side with muscle and bone involvement.

Treatment Plan. Via an oral approach, reduction of the hypertrophy of the masseter muscle by 50% of its vol-

ume and reduction of the bony surplus in the angle area, according to the technique shown in Fig. 97a.

Actual Treatment. This was performed as planned. The surgical correction resulted in a symmetrical facial appearance as seen on the full face photograph (Fig. 41c). The *p.a. cephalogram* 18 months later supported the skeletal background of the clinical picture.

Discussion and Conclusion. A unilateral masseter hypertrophy can, to a certain extent, simulate a unilateral H.H. However, there are, clinically as well as radiologically, clearly distinguishable differences. There is no irregular enlargement of the condyle, no downward convexity of the lower border and no typical rounding of the angle; on the contrary there is a surplus of masseter musculature and often also prominence of the bony angle area. The differential diagnosis is simple, as the classic bony signs of H.H. are absent on the radiographs.

12.3

Bilateral Hemimandibular Hyperplasia

If it is true that these two postulated condylar growth regulators exist, it has to be expected that the M-factor of both sides, left and right, can become hyperactive. In these cases a similar picture of H.H. will appear on each side, sometimes almost symmetrically. The facial asymmetry would be much less evident than in the unilateral form. The bilateral inferior border convexity meets in the chin region thus producing a very strong mandible

Questionable Bilateral H.H. (Fig. 42)

Sex and Age. Male, 31 years of age.

Aetiology and History. One year previously the patient had noticed that his left mandible gradually had become larger than the right and that his chin had moved over to the right side. He wanted to know whether or not this growth abnormality would continue and what could have been done to stop it. He had always had a strong mandible.

Clinical Findings. The face of this patient looks asymmetrical from in front, the left side being longer than the right and the right angle region more laterally than the left and also more than normal (Fig. 42a–e). In profile view both sides seem longer than normal, the left side more than the right. The left angle is more inferiorly situated than on the right and it is tilted medially compared with normal. There is a slight downward convexity of the lower border of the left body. The lip commissure is slightly oblique with its left corner slightly lower than the right. The right angle is very pronounced, well rounded and laterally tilted. Its lower border seems almost horizontal, but still with a slight inferior convexity which ends in the chin region. The chin prominence deviates from the facial midline to the right side by a few millimetres.

The class I occlusion (Fig. 42f), with a medium degree of deep overbite, is slightly twisted with the teeth on the left side more vertical than on the right. Due to a slight lingual tilting they seem shorter. The lower dental midline is over to the right by 2 mm and also the right lower front teeth are tilted towards the facial midline. There is an oblique occlusal plane of minor degree with the right side higher than the left. The facial nerves and the mouth opening were unremarkable.

Radiographs. The panoramic view of the mandible (Fig. 42g) discloses a rather interesting and unusual shape of the lower jaw: there are two large condyles. The left condyle is larger than the right and shows an anterior beak-like deformity. The right condyle is almost

similar to that seen in a case of acromegaly. However, the radiograph must show the rather typical enlargement of both condyles, the increase in body height and the inferior border convexity. Also coarse trabeculation should be found, and depending on the speed at which the deformity developed, there will be inferior displacement of the mandibular canal. For me, one very important point would be that the bilateral form should not be symmetrical. In bilateral H.E. we always expect to see asymmetry and that is also true for bilateral H.H.

round, but clearly larger than normal. It also has the beginning of a “beak” on the anterior surface. Both necks seem thicker than normal and the right clearly longer than the left. Both ascending rami are rather long, the left longer than the right and both angles are very rounded with a larger radius on the right than the left. Both lower borders are moderately but clearly downwardly convex, meeting at the symphysis at a higher level than the lower borders.

Compared with the normal, there is an increase bilaterally in the vertical height of both mandibular bodies, on the left slightly more so than on the right side. The trabecular structure, as far as is visible, seems rather coarse on both sides. The mandibular canal on both sides runs at a greater distance from the apices of the molars and bicusps than normal, the left more than the right. But neither is really displaced close to the lower border. On the left side there is a clearly defined translucency in front of the angle, close to the lower border. This is regularly found in that shape and location in cases of a pseudo-cyst due to indentation by a part of the submandibular gland.

The tomograms of the condyles and rami (Fig. 42h, i) show, on the right side, an unusually large but normally shaped condyle and neck while the left condyle has the typical shape of a H.H. deformity, though not overly impressively so.

The mandible in open p.a. projection (Fig. 42j) permits one to see clearly the coarse trabeculation, particularly in both ascending rami and in the very large condyles. The left ascending ramus is clearly longer than the right. Both horizontal rami are very high and have a distinct coarse trabeculation. They look rounded at their lower borders and form a very pronounced notch at the symphysis.

The cephalometric view in p.a. projection (Fig. 42k) also demonstrates the vertical enlargement of both horizontal rami with the very clear convexity of their lower borders and the symphysis positioned remarkably to

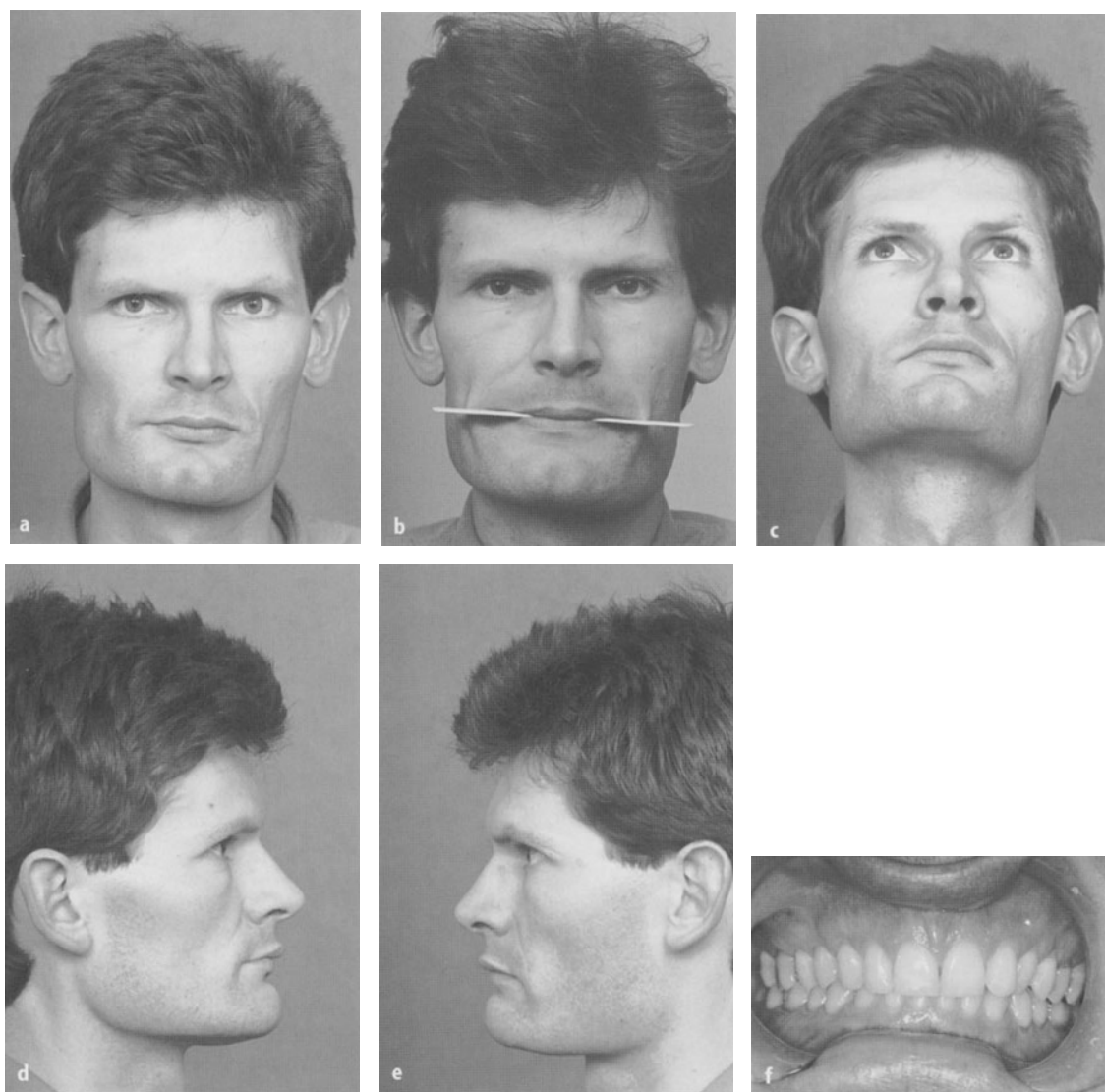


Fig. 42a–f. Questionable bilateral H.H. **a–e** The patient's appearance from in front, from below and from both sides and also with a spatula demonstrating a mild degree of obliquity of the occlusal plane shows all signs of a possible bilateral H.H. **f** The occlusion shows the typical picture of an unilateral H.H. with teeth tilted towards the more pronounced side and the necessary height difference between the two premolar-molar areas. There is a slight deep overbite

the right of the facial midline. It also shows that on the left side the maxillary sinus extends more inferiorly than on the right.

The evaluation of the tracing of the panoramic radiograph (Fig. 42l) discloses on both sides the measurements consistent with a H.H., on the left even more than on the right side. In addition, clear differences in length of the ascending and horizontal rami are noted. The left horizontal ramus is 24 mm longer than the right and its trunk measures 9 mm more in length than the right. The left maxillary sinus reaches 3 mm further inferiorly

than does the right and this is also true for the left maxilla. The difference in body height in the molar regions measures 6 mm more on the left side than on the right, which is also rather high, measuring 38 mm. Both angles are much smaller than average.

The requested *skull scintigraphy* was performed with 800 MBq Tc-99m-DPD. They were evaluated as maximum 100% in the right condyle and 153% in the left condyle. This proved that in the left condyle a very remarkable hyperactivity existed (Fig. 42m).

Provisional Diagnosis. Most probably bilateral H.H.; on the right side the growth was inactive and on the left there was a clear hyperactivity indicating growth factor M-type production, and also, to a small extent, factor L production.

Treatment Plan. High intracapsular condylectomy on the left side via a retrotragal approach, and, via an intra-oral approach, reduction in the surplus height of the left mandibular body and angle. In addition, the prominence of the chin should be shifted to the left and slices of lyophilized cartilage placed on the left lateral aspect of the ascending ramus and body for correction of the unaesthetic flatness of the left lower half of the face.

Actual Treatment. The surgery was carried out as planned by one of my trainees. As a result of the surgery, the for-

merly tilted teeth on the right side came into an upright position within only 4 months without any surgical or orthodontic occlusal correction. The symmetrization of the mandible had produced quite a pleasing result.

Discussion. This was not an easy case to diagnose properly. There was no doubt that on the left side there was a very active H.H., which only started when the patient was about 30 years of age. There was also a minor degree of L-factor hyperactivity, because the symphysis and the chin and the midline of the lower teeth were shifted slightly to the right side. The measurements of the tracing revealed that both the horizontal as well as the ascending ramus were remarkably longer on the left side than on the right. So, we must diagnose the left anomaly as a clear hybrid form with the M-growth regulator predominant.

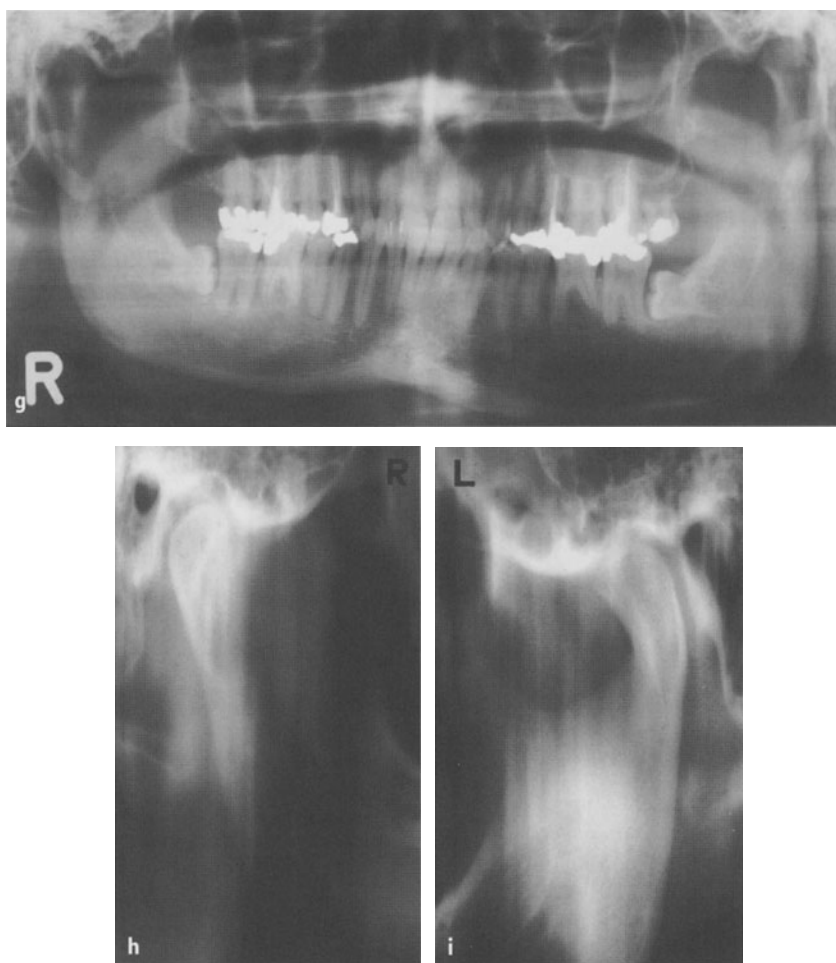


Fig. 42g-i. Questionable bilateral H.H. **g** The panoramic radiograph shows on the right side of the mandible all the characteristic signs of a H.H. and even more so on the left. There is a rounded translucency at the left angle, typical of a so-called pseudo-cyst caused by the impression of an enlarged lobe of the submandibular gland. **h, i** The tomograms of the mandible in the p.a. projection in the open mouth position show, on the right side, a very large condyle of probably normal shape and on the left the characteristic enlargement and deformity of a H.H.

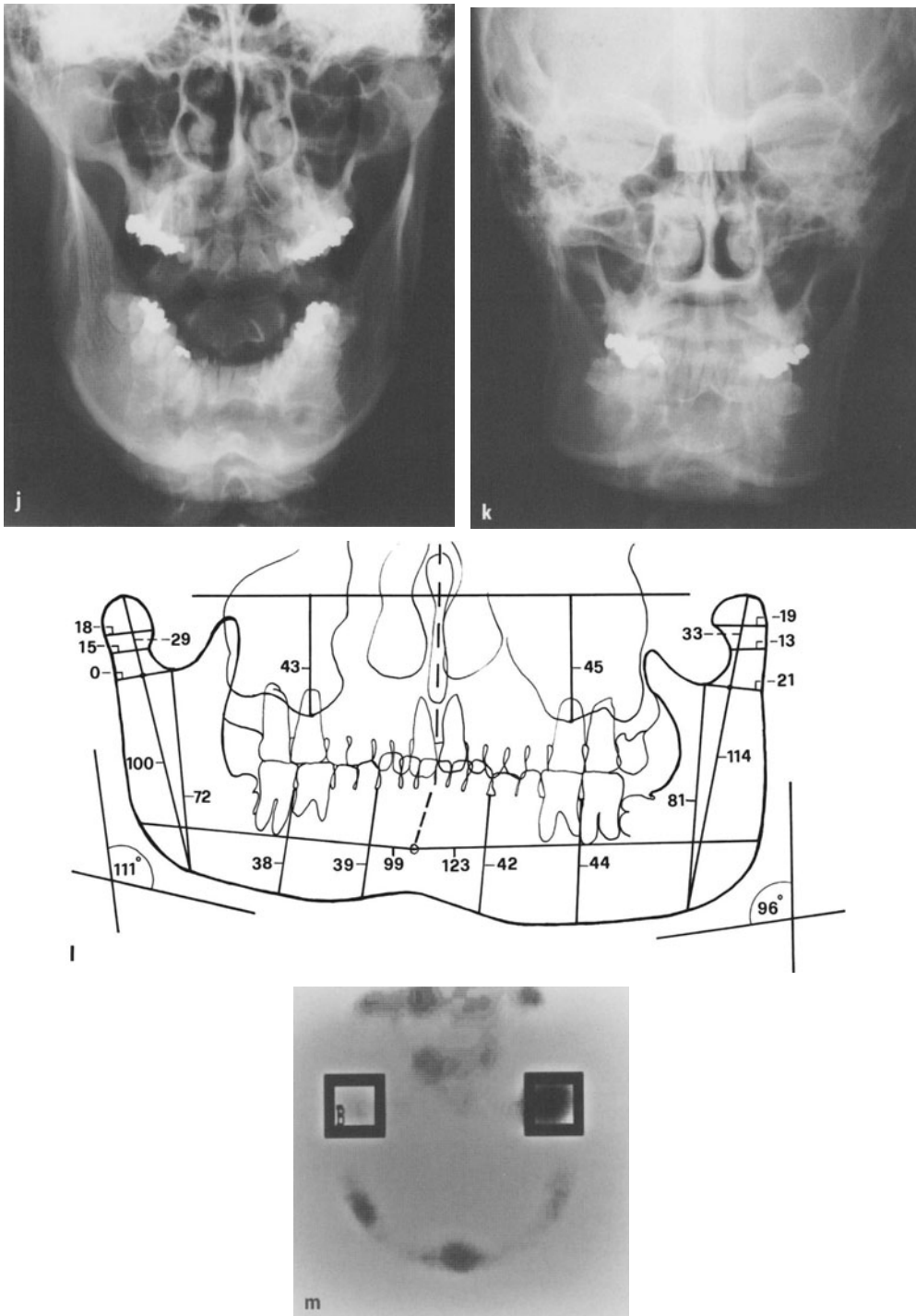


Fig. 42j-m. Questionable bilateral H.H. **j** The radiograph of the mandible in p.a. projection in the open mouth position, shows that both condyles are very large and both rami are very thick, the left one being longer than the right. The typical strong coarse trabeculation of both sides cannot be overlooked. **k** The p.a. cephalogram shows, on both sides, the typical downward convexity of the inferior borders of both horizontal rami and all other signs of a H.H. anomaly, the left even more pronounced than the right, including the coarse trabeculation. **l** The measurements of the tracing of the panoramic view indicate clearly a left side hybrid form of H.H. and H.E. with the H.H. type predominant on top of a possible bilateral H.H. **m** The scintigram reveals very pronounced hyperactivity in the left condyle but no activity in the right

The question arose whether or not the shape of the right mandible was also due to a now inactive H.H. or due to the deep overbite mandibular deformity alone. The clearly existing height increase in both mandibular bodies and the convexity of the lower border on the right side with the very pronounced notch in the symphysis region suggested the existence of a H.H. on the right side too. Also, the very large condyle and thick neck and the massive right ascending ramus supported that opinion. The very pronounced coarse trabeculation in the whole mandible including both condyles, also suggested to me the existence of H.H., as this is not seen in similar mandibular deformities of different origin. The fact that the mandibular canal was not displaced to the lower border was not a contradictory argument. We have seen this happening in the case of Fig. 33, in which

the H.H. developed very gently and slowly for over 25 years.

There was another interesting fact to be mentioned. Not only according to the patient's history did the asymmetry start to become visible at the age of 30 years, but it also started to be active again, most probably superimposed on an already existing bilateral inactive H.H. deformity. But in spite of all signs of a possible bilateral H.H. there is no absolute proof.

Conclusion. This case seemed to prove the possibility that a slow growing bilateral H.H. can occur almost symmetrically and that even at the age of 30 years, a superimposed more hyperactive phase can develop.

It also permits the conclusion that there are cases which are very difficult to diagnose with certainty.

12.3.1

Differential Diagnosis of Bilateral H.H.

Naturally there is much less possibility of a pseudo-bilateral H.H. than of the unilateral one. But there are two diagnostically very difficult possibilities. One is bilateral masseter hypertrophy and the other is the mandibular shape in cases of class II occlusion with severe deep overbite. These two anlage-induced anomalies can look very similar to a bilateral H.H. The deep overbite case is always very symmetrical and therefore not very likely to be a bilateral H.H. case. Bilateral masseter hypertrophy is often asymmetrical and can also produce diagnostic difficulty. There are two pronounced differences which we will always have to find: one is the increase in body height in the premolar-molar regions with the downward curvature of the lower border of the mandible and the other is the enlarged condyle and neck.

There is also an additional differential diagnostic aid. Scintigraphic examination may still prove that both sides are hyperactive, but one side more so than the other, or even one side only.

There is still another possibility of making a wrong diagnosis: the enlargement of the mandible in acromegaly cases. However, that abnormality of the mandible has to be symmetrical as it is caused by a central steering mechanism. Acromegalics may have quite enlarged condyles but they will also have the other typical features of the disease such as enlargement of the supraorbital rims, of the paranasal sinuses, of the fingers and toes and the typically thickened skin.

Bilateral Pseudo-H.H. in a Case with Class II Occlusion with Deep Overbite (Fig. 43)

Sex and Age. Female, 18 years of age.

Aetiology and History. The patient's mother had a similar jaw appearance. The patient was referred for surgical correction of her retromandibulism.

Clinical Findings. Typical class II appearance (Fig. 43a, b) with deep overbite with a pronounced labio-mental fold, a well-pronounced chin and almost rectangular angle areas, with strong masseter muscles on both sides and well-defined mandibular lower borders.

The occlusion (Fig. 43c–e) is also very typical of that type of anlage-induced mandibular anomaly: the upper front teeth cover the lower ones completely. The fully erupted crowns of the upper and lower molars and bi-

cusps are very short in height, and are in a class II occlusal relationship.

Radiographs. The panoramic view (Fig. 43f) shows a very symmetrical shape of the two hemimandibles. Both condyles are equal in size and shape, rather large, but within normal limits. They both seem to have a slight anterior "beak". The ascending rami are broad and normal in height. Both mandibular angles are very rounded with a rather large radius. They are almost at right angles to the horizontal part of the mandible. There is no real downward convexity of the lower border and no increase in body height in the molar-bicus-

pid regions. But in the front region there is a remarkable reduction in anterior mandibular height compared with normal. Due to the normal height of the horizontal rami

in their lateral parts and the lack of height in the front region there appears to be a pseudo-downward convexity of the lower borders which can give rise to misdiag-

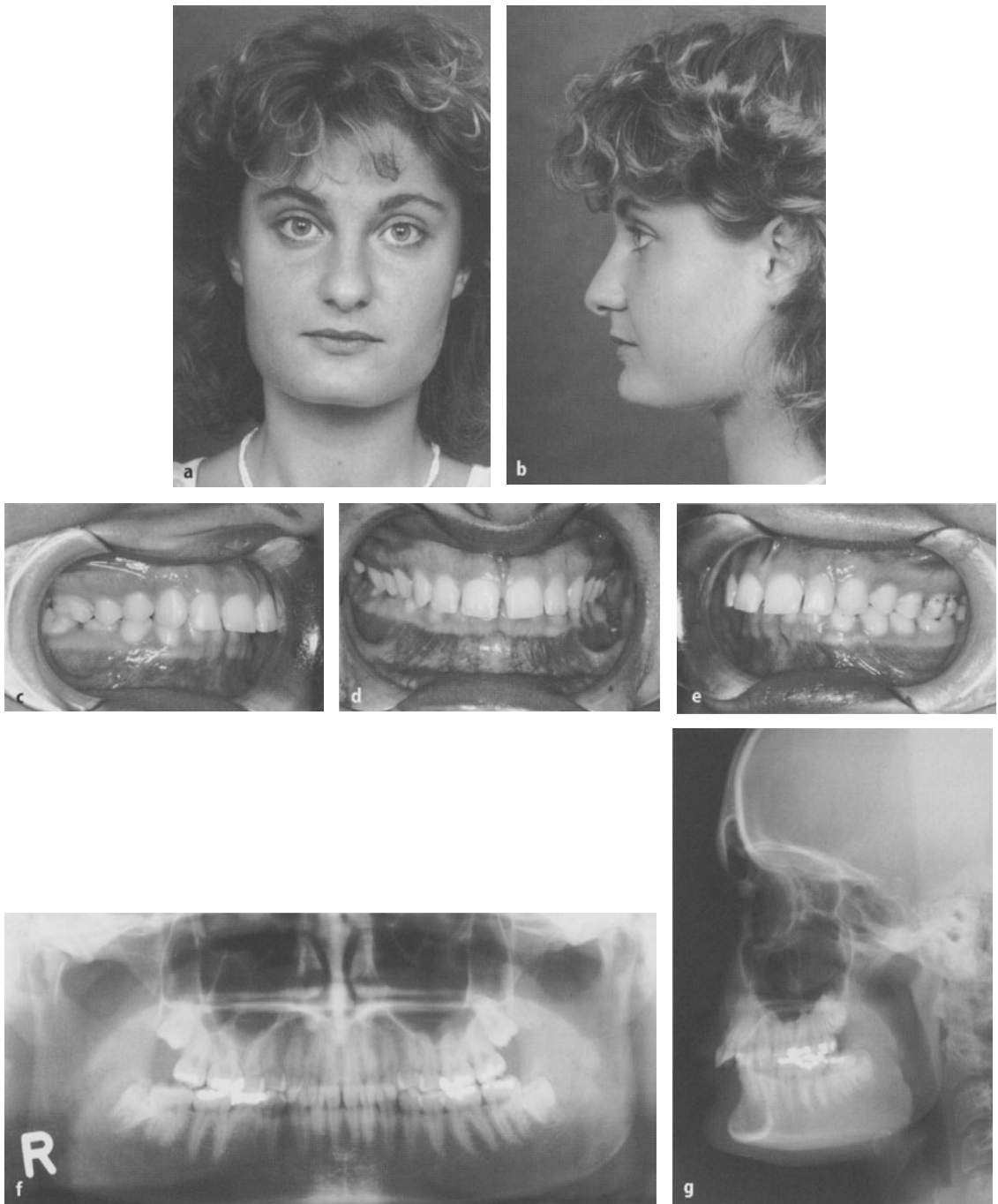


Fig. 43a–g. Bilateral pseudo-H.H. in a case with class II occlusion with deep overbite. The patient's appearance from **a** in front and **b** the profile view, typical of a retromandibulism case with a deep overbite. **c–e** The occlusal situation is also very typical for such an anlage-induced mandibular anomaly. **f** The panoramic view discloses a similarity with a possible bilateral symmetrical H.H., but several important signs of H.H. are missing. **g** The lateral cephalogram discloses the typical appearance of a retromandibulism-deep overbite deformity

nosis as a bilateral H.H. The mandibular canals run rather close to the apices of the third molars and they are not visible further anteriorly. Both halves matched at the symphysis, without a real kink or notch, compared with that seen in the case of Fig. 42.7. The trabeculation structure appears normal in all parts of the mandible on both sides, including the condyles.

The lateral cephalogram (Fig. 43g) shows the classic picture of a retromandibulism with deep overbite, in particular with a strong mandible which lacks height in the chin region compared with the normal. There is no downward convexity of the lower borders.

The measurements of the panoramic radiograph reveal two almost equal halves of the mandible.

Provisional Diagnosis. Retromandibulism with deep overbite and hypogenia, simulating a symmetrical bilateral H.H.

Treatment Plan. After presurgical orthodontics, surgical advancement of the mandible and vertical enlargement of the chin using a sandwich technique.

Bilateral Pseudo-H.H. in a Case of Bilateral Masseter Hypertrophy (Fig. 44)

Sex and Age. Female, 35 years of age.

Aetiology and History. During puberty, both mandibular angle regions had become larger and more prominent than normal, in particular the angle on the left. The patient came for correction for aesthetic reasons. Nothing relevant in the family history.

Clinical Findings. The face appears almost rectangular when looked at from in front. Both angle areas are much more voluminous than normal, the left side a bit more than on the right (Fig. 44a–c). Looking at the patient from below there seems to be a certain degree of downward convexity on both sides, again more so on the left than on the right. However, this inferior convexity is located in the angle region and not in the mandibular body. When closing the teeth forcibly, the masseter muscles become even more prominent than normal, again especially on the left side. Simultaneously the temporalis muscles also become much more prominent than normally observed (Fig. 44d). Mouth opening was limited to 33 mm intercuspal distance. On palpation, the bulk of the angle regions was mainly the musculature, with strong bony angle contours underneath. Innervation of the facial musculature and the skin was normal. The occlusion was perfect. There was no deep overbite nor any occlusal asymmetry.

Actual Treatment. The patient refused any surgery.

Discussion and Conclusion. A typical retromandibulism with deep overbite and hypogenia can clinically and radiologically appear very similar to a bilateral symmetrical H.H. A few signs of the latter were missing. There was complete symmetry. Since H.H. has its origin within the condyle, influencing that respective hemimandible only, therefore it could hardly ever appear absolutely symmetrical. For symmetrical hyperactivity of the condylar growth factors a higher steering mechanism would have to be the cause, as in acromegaly.

Another very important absent sign was the fact that the distance between the apices of the cheek teeth and the lower border of the mandible was not increased. Therefore, the very typical inferior convexity was also missing. And thirdly there were no signs of any coarse trabeculation in all sections of the two halves of the mandible. So the whole could only be termed bilateral pseudo-H.H.

Radiographs. In the panoramic view (Fig. 44e) the mandible has an almost rectangular shape with condyles of normal size and form. Both angles are rounded but have a slight antegonial depression in front of them, more pronounced on the left than on the right side. Both angles seem to have a rather strong protuberance, the left more than the right. The trabecular structure of the mandible and the condyles is unremarkable. The distance from the apices of the molars and bicuspid to the lower border of the mandible is within the normal range and there is not the slightest inferior convexity of the lower border in that region. The mandibular canal seems to run fairly close to the inferior mandibular cortical margin.

The mandible p.a. projection in closed position reveals that in both angle regions there exists a flaring, on the left side more so than on the right.

Provisional Diagnosis. Severe bilateral masseter muscle hypertrophy and symmetrical hypertrophy of the temporalis muscles on both sides.

Treatment Plan. Transoral reduction of both masseter muscles and angles according to our standard procedure (see Fig. 100a).

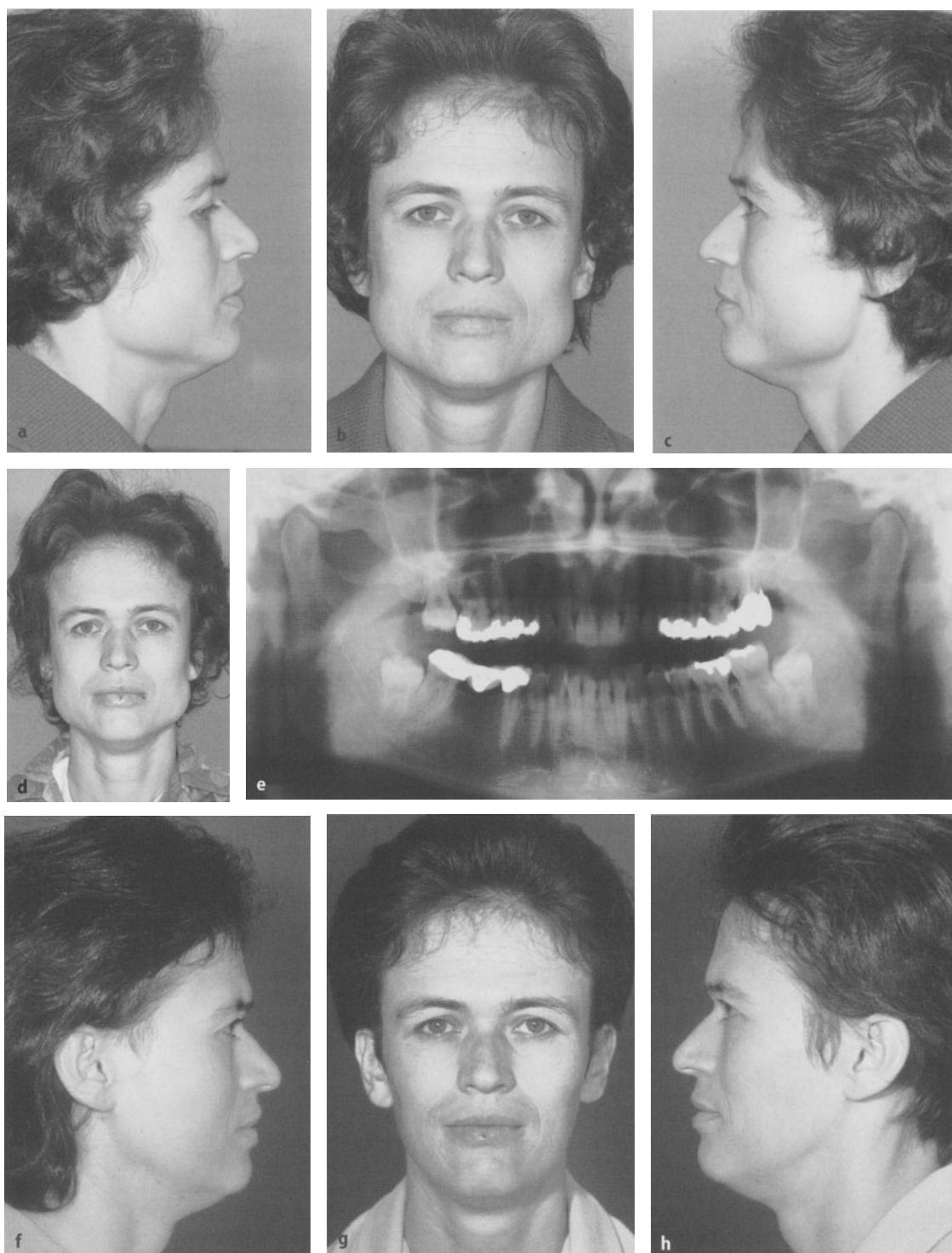


Fig. 44a–h. Bilateral pseudo H.H. in a case of bilateral symmetrical masseter hypertrophy. **a–c** The patient's appearance from in front and both profile views. **d** When closing the teeth forcibly, the masseter muscles as well as the temporalis muscles become very disturbingly ugly. **e** The panoramic radiograph discloses a mandible within the normal shape and structure except for very strong angles. **f–h.** Bilateral pseudo H.H. in a case of bilateral symmetrical masseter hypertrophy. **f–h** The patient's appearance from in front and both profile sides, 1 year after surgical correction

Actual Treatment and Result. The correction was carried out as planned. Nothing was done to the hypertrophic temporalis muscles. They were covered by the hair. Normal mouth opening was achieved during surgery by force, and postoperatively through the patient's brave exercising. It resulted in a symmetrical slim face with unobtrusive angle areas (Fig. 44f-h).

Discussion and Conclusion. A bilateral masseter hypertrophy case can have quite an amount of surplus of bone in the angle region which will produce an inferior convexity in the angle region only. However, it could hardly be mistaken for a bilateral H.H. as there were no enlarged condyles, and, what was even more important, the inferior convexity was never in the molar-premolar areas. This would have to exist even in a very mild degree of bilateral H.H. in which the condyles and their necks are not clearly enlarged.

Hemimandibular Elongation (H.E.)

13.1

Main Clinical Features

Hemimandibular elongation is the second form of misregulation of mandibular growth due to condylar hyperactivity. We believe it is due to hyperactivity of our assumed growth regulator L. It exists in unilateral as well as bilateral forms.

This growth anomaly of the mandible differs entirely in its clinical and radiological picture from hemimandibular hyperplasia. Its main characteristic feature is not bone production in mass as in H.H., but elongation of the affected hemimandible, in all its sections (Fig. 46). As a consequence, the most striking features are deviation of the chin and of the midline of the lower teeth to the opposite side and a crossbite on the normal side. There is no height difference between the two sides of the face, contrary to H.H.

13.2

Unilateral Hemimandibular Elongation

In the following, I will discuss the unilateral form only. We distinguish two types of H.E., the slender and the non-slender type. There are also cases which show a picture in between the two. Why the L regulator can produce two distinguishable types of H.E., the slender and the non-slender, we do not know. Both produce the same two main features: elongation of the affected hemimandible with a crossbite and chin deviation to the normal side. The *slender type* always has a clearly stretched mandibular angle while the *non-slender type* may have an almost normal facial configuration except for the crossbite and the chin deviation, almost without any effect on the position of the teeth. A slight tilting of the lower front teeth towards the elongated side is mostly found. In the average case of H.E. the mandible just seems to be pushed horizontally over to the opposite side. Otherwise the alignment of the arches of teeth of both jaws is absolutely normal. Only when the growth push is very strong and rapid does it push not only the mandible over to the normal side but it also produces an open bite, either on the affected or the normal side. In all cases the lower dental midline is out of the facial midline.

When the mouth is open, the mandibular asymmetry may not be recognizable in mild cases, but in severe cases the mandible remains asymmetrical even on maximum opening.

On scintigraphy, H.E. will show the same behaviour as the H.H. type, but with a much smaller condyle. During the normal growth period the diagnosis of hyperactivity cannot be made by comparing the findings with the contralateral side. The existence of hypoactivity on one side and physiological activity on the other side could be wrongly diagnosed as hyperactivity. In such a case, the diagnosis of hyper- and hypoactivity can be made according to the RU of Tc-99 m-MPD only, by comparing the result with the figures published by Kaban et al. (1995), shown in Fig. 9.

13.2.1

Nomenclature of H.E.

Many authors include H.E. in the generally used term “condylar hyperplasia”. But this anomaly never does have, neither clinically nor radiologically, an enlarged condyle. So it is definitely wrong to call it a condylar hyperplasia. It is, of course, the result of condylar hyperactivity, specifically of our assumed condylar growth factor L. H.E. is commonly called “unilateral mandibular prognathism” or “deviation prognathism” (R. Bruce and J. Hayward 1968). This is definitely wrong, because the unilateral H.E. patient is, in general, not prognathic. This is easily recognizable in the lateral cephalogram. In spite of the unilateral elongation, the mandible deviates to the opposite side only, but does not project forwards, except in bilateral H.E. cases or when the unilateral H.E. occurs in an ordinary antemandibulism case. The terms “unilateral skeletal macrognathia” (N. Rowe 1960) or “unilateral transverse hypercondylism” (A. Gordeff et al. 1988) or “rotational condylar hyperplasia” (L. Kaban 1988a) are also used.

I feel that none of these frequently suggested names for the H.E. anomaly are consistent with the anatomical facts. As in all these cases of unilateral H.E., all parts of the mandible (horizontal and ascending rami, condyle and condylar neck) may be longer than those of the contralateral hemimandible. In any cases of H.E. the affected side as a whole is longer than the normal side.

Therefore, I prefer to call it unilateral or bilateral hemimandibular elongation. As previously mentioned, with-in this anomaly we have to distinguish between a slender and a non-slender type. This distinction can often not be made by the clinical appearance of the case but only on the radiographs. So, we can speak about a H.E. slender and a non-slender form, and a medium slender form, or a slender/non-slender form.

13.2.2

Radiographic Findings in Unilateral H.E.

Panoramic Projection in the Closed Teeth Position

This gives very important information about the chin and the lower teeth midline position in relation to the facial midline. That is generally clearly definable by the nasal crest of the palate and the nasal septum. It also permits comparison of the two hemimandibles, including their angles and condyles together with their necks.

Slender Type. The slender type always has, not only an elongated hemimandible on the affected side, but also a stretched angle. The elongation involves the horizontal and the ascending ramus and the condylar neck. At its upper end there is generally no clear distinction between it and the condyle. The condyle looks rather like a slight thickening at the upper end of the neck, the horizontal ramus has less height than a normal mandible. The mandibular canal is clearly visible and runs close to the apices of the roots of the teeth. The trabecular structure is unremarkable.

Non-slender Type. The non-slender type looks almost like a normal half of the mandible and yet it is clearly elongated as a whole, but not so clearly in all its sections as seen in the slender type. The condyle seems almost normal in shape and size. The difference in length compared with the normal side is easily demonstrated when all its parts are measured exactly. In this non-slender form, one sometimes gets the impression that the condylar neck might be slightly thicker than normal. Even the condyle itself seems slightly enlarged. The horizontal ramus is not as slim as in the slender form. Instead of the clear stretching of the angle there is only a certain degree of reduction of angle contour, which seems to lead to a slight stretching compared with the normal side. There is not that striking reduction in height of the horizontal ramus that is generally found in the slender type. But the two main features of a H.E. growth abnormality are always present: chin asymmetry and cross-bite on the normal side without increase on facial height.

Mild Slender or Slender/Non-slender Type. Only in the very clear extremes can one clearly distinguish between a slender or a non-slender type of H.E. It seems almost self-evident that a high percentage of the H.E. cases lie somewhere inbetween the slender and the non-slender types. This is diagnosable on X-rays only.

We can imagine that in those non-slender cases and also in the cases of a slender/non-slender type, rudimentary influence of the M-regulator also exists.

TMJ Tomographs. These disclose clearly the difference in shape and length of the condylar process (condyle and its neck). The head looks almost like an elongation of its neck, not clearly distinguishable from the neck. If the ramus-angle area is also included in the TMJ tomograms then the amount of angle stretching becomes obvious.

Mandible in p.a.-Projection in the Maximum Open Mouth Position. This projection shows better than any other conventional radiograph the difference in length of the rami of the two sides and also the deviation of the chin prominence and the lower teeth midline over to the normal side. It also gives evidence regarding its structure.

Paranasal Sinuses, Water's View. Although this radiograph does not specifically deal with the mandible, it is often of great help in determining the facial midline in comparison with the dental midline of the upper jaw as it shows both the crista galli as well as the upper teeth. Once we are sure about the location of the upper teeth midline then we can also correctly determine the lower teeth midline in relation to the facial midline.

This radiograph also gives an overall view of the size, extent and pathology of the maxillary sinuses. It is often important to know this before surgery.

Scintigraphy will reveal whether condylar growth activity is still going on or not. As mentioned earlier, the scintigraphic findings in the condyles look very much the same in a case of real unilateral hyperactivity or in a case of unilateral hypoactivity during the normal growth period. In such a situation, the hypoactivity side looks inactive while the other side shows normal growth activity, but which is easily misdiagnosed as hyperactivity. Therefore, we must always be sure that condylar hypoactivity does not exist. This can be misinterpreted as a contralateral H.E. A wrist radiograph may help to determine whether or not normal physiological growth is still going on. The RU figures should help to make a correct diagnosis in such a case as the RU findings can be compared with the normal findings of the patient's age group.

In the following, some cases will demonstrate the variations in unilateral H.E.

13.2.3

Treatment Guidelines for Uni- and Bilateral H.E.

As mentioned in the Chap. 11.7 “Condylar Hyperactivity” under “General Surgical Treatment Considerations”, the philosophy in treating the H.E. cases differs quite a bit from that for H.H. and hybrid form cases. Only in a very rapidly growing situation is early high condylectomy indicated (see Fig. 28). Otherwise we can wait until the final position of the maxilla is clear.

In many cases I found that the mandible could be brought into proper occlusion without any presurgical orthodontics. It was as if the mandible, being primarily in proper occlusion with the maxillary teeth, has been pushed laterally by the abnormal growth due to H.E. Naturally, there are also cases in which the dental arches will have to be arranged by presurgical orthodontics to be ready for the surgery when no further growth of the mandible is to be expected. Then surgery can deal with the correction of the maxillo-mandibular complex as a whole. Too early surgical correction will lead to relapse as formulated in the principles: “*Growth can destroy your early good result*” (Principle No. 27) and “*Too early corrective surgery on the maxilla will result in reduction of its growth capacity and too early osteotomies of the mandible will end up with a relapse*” (Principle No. 28). The type of surgery indicated depends mainly on the surgeon’s imagination and ability. After growth

has ceased, a bilateral splitting procedure of the rami will be sufficient to correct the anomaly if the desired occlusion can be achieved. In some cases, even in very pronounced ones, I achieved a very acceptable result with an unilateral sagittal splitting procedure only, as did P. Mercier (1969), who had trained with me for some time. The necessary rotation in the joint of the non-operated side never caused any problems in these cases (see Fig. 45).

As mentioned in the introduction to this book, in many of the cases on which I had to operate, neither presurgical nor postsurgical orthodontics were available. In these cases the surgical correction had to overcome the occlusion difficulties by additional alveolar segmental osteotomies or by extracting a tooth and performing an ostectomy at its site. I found a parasymphysal ostectomy very useful in several cases of H.E., in unilateral as well as bilateral forms (see case Fig. 59). Naturally such an asymmetric movement of the two halves of the mandible had to be planned carefully in a model operation.

Whenever I did an ostectomy in the symphyseal region, I first cut off the chin prominence, leaving it muscle-pedicated on the digastric-geniohyoid muscles. This chin segment was then used as a bridging piece of bone below the vertical ostectomy line. With this procedure it was also possible to correct an existing chin asymmetry simultaneously.

Unilateral H.E., Slender Type, before Puberty (Fig. 45)

Sex and Age. Female, 14 years and 0 months.

Aetiology and History. The chin asymmetry was first noticed at the age of 9 years. No direct aetiological cause was found. An uncle and a great aunt reported that they both had a prognathic lower jaw. From the age of 10 years the girl was under constant orthodontic treatment. At that age a prosthesis with an expansion screw was worn for widening the maxilla and a monobloc and chin cap was worn by the girl very bravely, in order to stop further forward and lateral growth of the mandible. At 13 years of age the girl’s general growth was complete as was that of the mandible. The orthodontic appliances had not been able to influence the forward and lateral overgrowth of the mandible.

At exactly the age 14, the girl was referred for surgical correction for functional as well as psychological reasons. There were no TMJ pain problems, 42 years later the patient had three children and several grand children. None of them has a similar mandibular abnormality nor an antemandibulism.

Clinical Findings. The outer appearance is the typical picture which is often called unilateral prognathism or laterognathism (Fig. 45a). The lower jaw is shifted to the right side and protrudes in the manner typical of an asymmetric prognathism. The lower lip is in front of the upper and the left lip corner is further dorsally than the right. Mouth opening was undisturbed with much less asymmetry than in the closed position.

The occlusion (Fig. 45b) shows the picture of a class III overbite to the right side producing a severe cross-bite with a shift of the midline of the lower teeth to the opposite side by the width of one lower incisor. The lower incisors exhibit slight tilting to the left.

Radiographs. The panoramic view was not available in those days, nor did we then order tomograms of the TMJ including the ascending ramus. *The lateral oblique view of the left mandible* (Fig. 45c) discloses a rather obtuse angle and a long thin condylar neck. The condyle itself is not visible. *The p.a. projection of the mandible* in the closed mouth position (Fig. 45d) shows a clear elon-

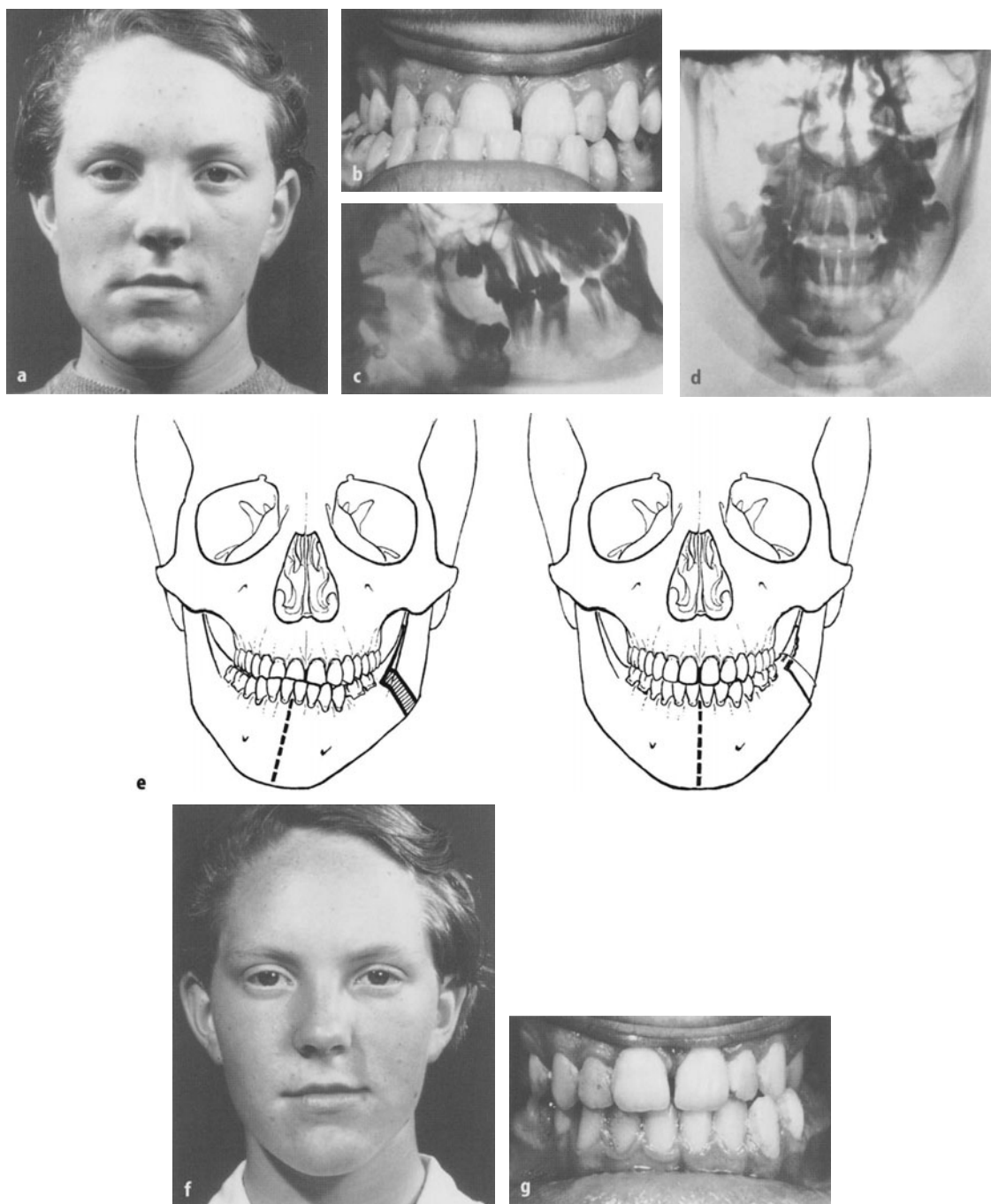


Fig. 45a–g. Unilateral H.E., slender type, before puberty. **a** A 14-year-old girl with unilateral H.E., typical appearance from in front, producing the picture of a “deviation prognathism”. **b** Presurgical occlusion: crossbite to the right side, already beginning in the left canine region. **c** Lateral oblique view of the left mandible. **d** The presurgical mandible p.a. projection in the closed mouth position discloses clearly the elongation of the left mandible. **e** Diagrammatic illustration of the unilateral sagittal splitting procedure performed. **f, g** The patient’s frontal appearance and occlusion 1 year after correction by a unilateral sagittal splitting of the ramus

gation of the left ascending ramus and articular process compared with the normal right side. The chin prominence is shifted over to the right side. The lateral cephalogram taken at that time has been mislaid.

Provisional Diagnosis. Severe unilateral H.E. of the left side, slender type.

Treatment Plan. As the girl's general growth and the abnormal mandibular growth had already come to a stop, it was decided (at the age of 14 years) to correct the situation by a sagittal splitting procedure on the rami followed by postsurgical orthodontic treatment if necessary.

Actual Treatment. First I performed my sagittal splitting procedure on the ascending ramus on the elongated side (Fig. 45e). When that left side was completely mobile the mandible could be rotated to the left into a very acceptable appearance and occlusion, bringing the

lower teeth midline and chin prominence into the facial midline (Fig. 45f, g). For that reason it was felt unnecessary to perform an additional splitting procedure on the right side. After intermaxillary fixation the ramus segments were fixed together in the new position by an anterior border wire, followed by 6 weeks of intermaxillary fixation. No additional postsurgical orthodontic treatment was instituted. *Forty-two years later* the facial symmetry achieved has not changed nor has the occlusion or the lower teeth midline. The relationship of the mandible to the maxilla is still without any signs of an antemandibulism. The panoramic view (Fig. 45h) of the mandible still discloses even then a typical slender form of a H.E. abnormality in the left mandibular half and the measurements of its tracing proves it (Fig. 45i).

There is still a certain degree of asymmetry of the mandible. The left side now has a rather short ascending ramus due to the corrective surgery using the sagittal ramus splitting procedure. The horizontal section of that side is still 10 mm longer than the opposite normal

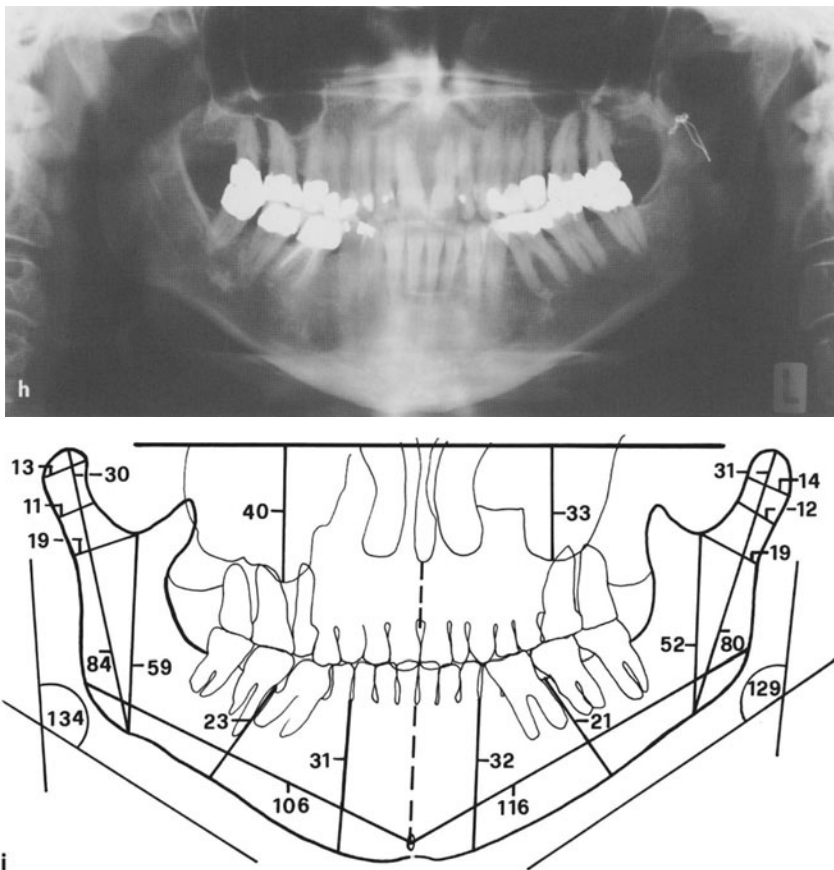


Fig. 45h, i. Unilateral H.E., slender type, before puberty. **h** The panoramic view taken 42 years after the unilateral sagittal splitting of the left ramus still permits the diagnosis of a former H.E. of the left side. **i** The tracing of that late panorama radiograph still proves the clear elongation of the left horizontal ramus

side and the angle is still stretched. Its condylar neck is strong and elongated. The midline of the lower teeth and the symphysis remain in the facial midline, unchanged. The p.a. projection of the mandible in maximum opening position now shows an almost symmetrical mandible, still with the elongated and stronger neck area on the formerly H.E. side.

Discussion and Conclusion. It is a great pity, that at the time when I operated upon that 14-year-old girl, there was no panoramic view of the jaws available. It would have helped us to know whether the H.E. was a real pure slender type of H.E. or already from the beginning rather

a slender/non-slender type as it now appeared. In spite of this, this case taught us a lot:

1. General skeletal growth as well as condylar hyperactivity can stop at the age of 13 years
2. The hyperactivity can produce such a strong abnormal growth of the affected side that a monobloc and chin cap can neither stop nor reduce it
3. The actual elongation may be unilateral only and can therefore be effectively corrected by surgery on the affected side only.
4. The typical elongated condylar neck may remain, so far for over 40 years, in spite of a set back operation on the mandible on the affected side only

Unilateral H.E., Slender Type of 10 Years Duration (Fig. 46)

Sex and Age. Female, 18 years of age.

Aetiology and History. The mandibular asymmetry became noticeable at the age of 7. The parents had no idea why it had started. As it gradually increased the patient's dentist started orthodontic treatment (at the age of 8 years). This went on for 10 years without any visible influence on the increasing asymmetry of the mandible. At the age of 18 she requested surgical correction, as a friend of hers with the same problem had had surgery with a very good result. There was no further increase in the asymmetry and change in the occlusion for almost 2 years.

Clinical Findings. The patient has a well-contoured, rather slim face with her chin shifted to the left side by about 5 mm (Fig. 46a). Her lip commissure is horizontal, but the left corner is a bit more lateral than the right. The right side of the mandible seems longer than the left. Mouth opening is normal, except that the asymmetry of the chin prominence becomes more obvious. The profile line is very harmonious (Fig. 46b), and of the vertical profile type, but the lateral shift of the mandible is easily recognizable. The function of the facial nerves was normal.

The occlusion shows a cross bite to the left side from the left first incisor distally (Fig. 46c–e). The midline of the lower teeth is shifted to the left side by more than the width of a lower incisor. Both lower and the upper right first molars are missing due to early extractions. The occlusal plane is absolutely horizontal.

Radiographs. The panoramic view (Fig. 46f) shows clearly the skeletal background of the asymmetry. The whole mandible is rather gracile. The left condyle is on the smallish side, but well-formed and has a rather short neck of normal width. The ascending ramus is also on the short side. In front of the well shaped angle is a

moderate antegonial notch. The right condyle seems slim. It has an elongated slim neck. It is an almost straight elongation of the posterior border of the ascending ramus, which is longer than that on the left. The right angle is well stretched. The right body is clearly longer than the left. Both are rather shallow in depth. There is a clear but small distance between the mandibular canal and the strong cortical plate of the lower border.

The TMJ tomograms (Fig. 46g, h) disclose a clear difference between the left and the right condyle and neck. The right condyle is not distinguishable from its clearly elongated straight neck. The left condyle is rather small with a superficial dense area. Its neck is short and seems rather thin.

The p.a. projection of the mandible at maximum opening position (Fig. 46i) shows the difference in length of the two hemimandibles very clearly. In this projection the right condyle is now distinguishable from the elongated neck. A clear deviation of the nasal septum exists which could indicate that the midline of the upper teeth is shifted to the left. But in fact the midline lies precisely in the midline of the base of the skull.

The lateral cephalogram (Fig. 46j) discloses no abnormality in the skeletal facial framework. The maxilla is of normal size and position as is the mandible. The profile line is of the vertical profile type.

The measurements of the tracing (Fig. 46k) of the panoramic view disclose clearly that the right side is elongated in all its sections: the right ascending ramus length is 75 mm, the left 65 mm. The length of the right articular process is 23 mm, the left 18 mm. The width of the right neck is 9 mm, the left 10 mm. The width of the right condyle is 10 mm and that of the left is 13 mm. The length of the right trunk is 51 mm and that on the left side 48 mm. The right horizontal ramus is 91 mm and

that on the left side 70 mm. In the molar region the height of the horizontal ramus is 25 mm compared to 20 mm in the same area on the left side. In the premolar-canine region the height is almost equal, 33 mm on the right side and 31 mm on the left. The two angles, on the other hand, differ remarkably. The right is clearly stretched (135°) compared with the normal and not only with that of the left side (125°).

Provisional Diagnosis. Unilateral H.E., of the right mandible, slender type and probably a very mild degree of contralateral hemimandibular hypoplasia.

Treatment Plan. Le Fort I-type osteotomy for slight advancement of the maxilla and bilateral sagittal splitting procedures on the rami. If necessary, additional correction of the chin asymmetry should be achieved by a sliding osteotomy (Fig. 46l).

Actual Treatment. The surgery was carried out according to the plan by M. Perko who was at that time a senior coworker in my clinic. An iliac crest marrow graft

was used in the maxilla as in most Le Fort I cases of those days. A chin-sliding osteotomy was performed in addition to the bilateral sagittal rami osteotomies. Wire osteosynthesis fixation was used for the maxilla and the chin. For the split mandible, circumferential wires held the segments in position and 6 weeks intermaxillary fixation guaranteed the bony union of the repositioned jaws in their new position (Fig. 46m–q).

Discussion. This case presented a typical, slender type, unilateral H.E. deformity of the mandible. Whether or not there also existed a minor degree of contralateral hemimandibular hypoplasia was very difficult to determine. The short left ramus and its rather pronounced antegonial notch might have suggested this. But the lack of an oblique occlusal plane due to reduced downward growth of the maxilla on the questionably hypoplastic side did not support such a diagnosis. However, it was very easily possible that the patient would have developed a retromandibulism with a class II occlusion if the H.E. on the other side had not overcorrected that situation.

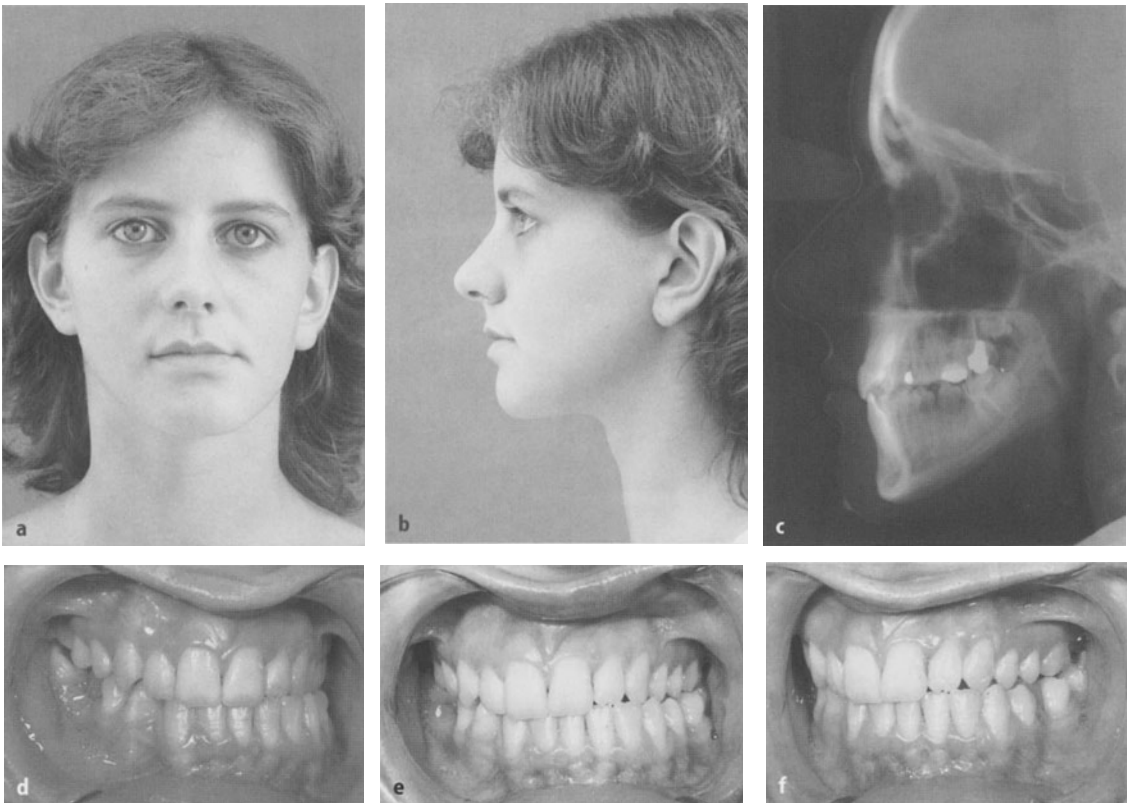


Fig. 46a–f. Unilateral H.E., slender type, of 10-year duration. **a, b** The patient's appearance from in front and profile are typical of this type of condylar growth L-anomaly with the elongated half of the affected side and deviation of the chin to the other side. **c–e** The occlusion is also very typical of this type of growth anomaly, a shift of the lower teeth midline to the left normal side producing a cross-bite on that side. **f** The panoramic view shows also a deformity of the mandible, very typical of this type of anomaly

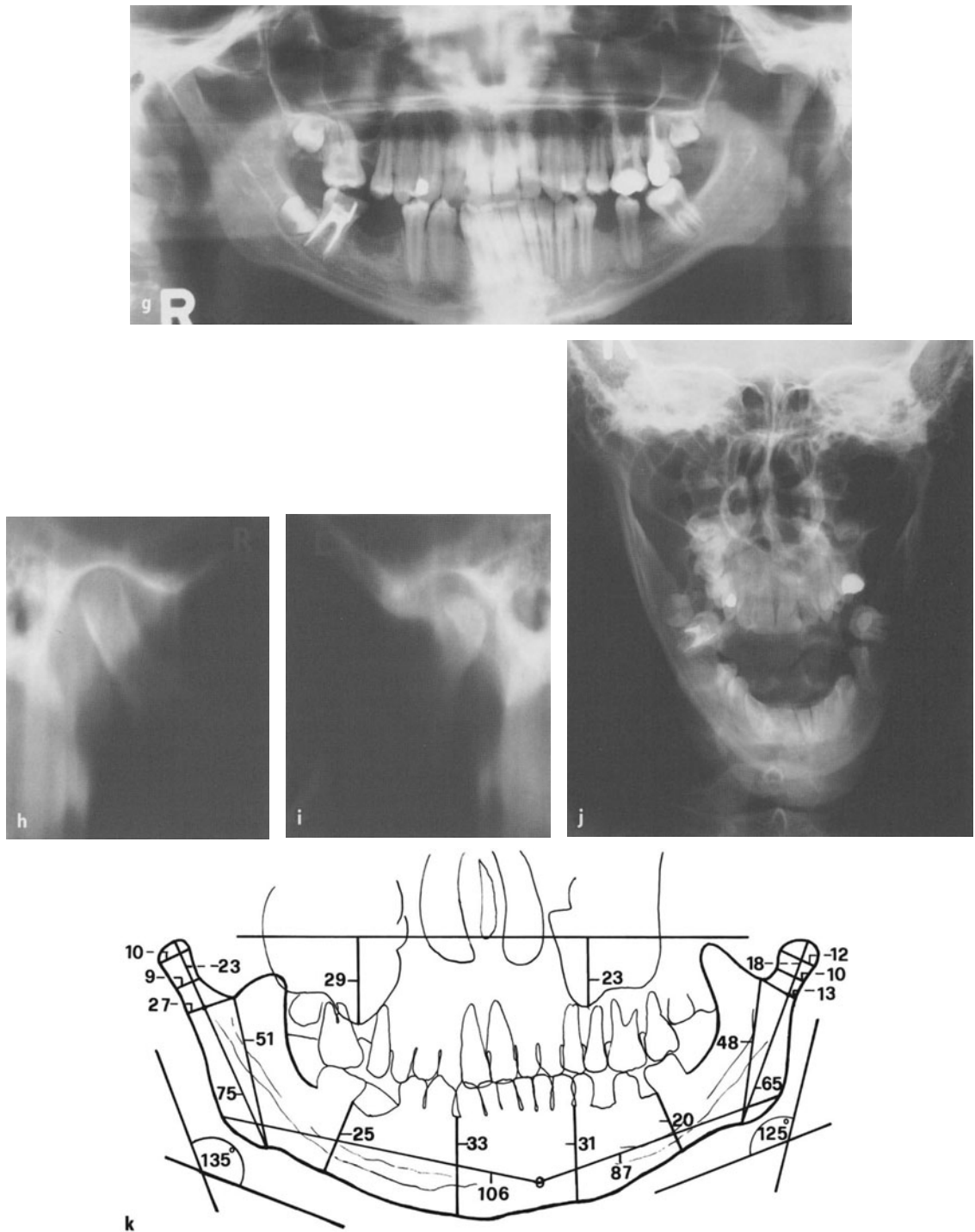


Fig. 46g-k. Unilateral H.E., slender type, of 10-year duration. **g, h** The TMJ tomograms show clearly the difference in the shape of the two sides. If they had included the ascending rami and angles also, the difference would have been visualized much better. **i, j** The p.a. projection of the mandible in the open mouth position discloses the enormous elongation of the right mandible even better than the panoramic view, while the lateral cephalogram does not show any asymmetry or antemandibulism-like position of the mandible. **k** The tracing of the panoramic radiograph shows the actual differences between the various sections of the two hemimandibles

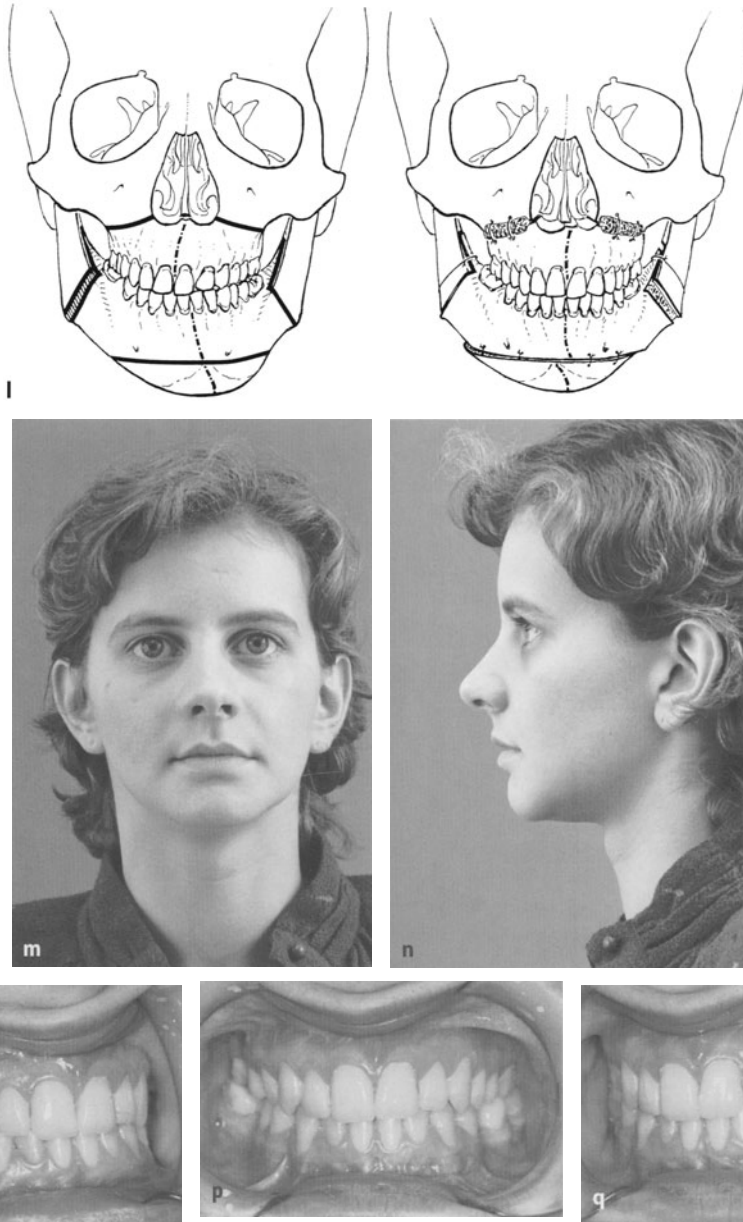


Fig. 461-q. Unilateral H.E., slender type, of 10-year duration. **I** Diagrammatic illustration of the planned corrective surgery. **m-q** The photographs, taken 1 year after surgery, show a perfectly symmetrical face and occlusion without any orthodontic treatment

Measuring the various sections of the two hemimandibles gave clear evidence that all sections were elongated on the right side as postulated for the unilateral H.E., slender type.

Conclusion. A typical slender type of unilateral H.E. was easy to diagnose on the radiographs. If the angle of

the mandible was very flat then that diagnosis might have been presumed clinically, without radiographs. However, standard radiographs are mandatory for a proper diagnosis, in particular as an unilateral hemimandibular hypoplasia might simulate the existence of a unilateral H.E. of the other side.

Unilateral H.E., Medium Slender Type (Fig. 47)

Sex and Age. Male, 20 years of age.

Aetiology and History. Within the last 5 years an asymmetry of the chin had developed without any known cause. The patient believed that there had been no further lateral growth of the chin in the past few months. Because of unavailability of scintigraphy in those days, he was advised to wait for 1 year more. During that year there had been no increase in the asymmetry as shown on photographs of the face and in the occlusion, and also as verified on the models.

Clinical Findings. There is a clearly visible facial asymmetry (Fig. 47a–c) with the chin over to the left side by 12 mm, with an elongation of the right mandible which seems more prominent laterally on the right side than on the left. It produces a more voluminous cheek contour compared with the rather flat cheek on the left side.

On palpation there seemed to be a greater volume of the masseter muscle and soft tissues in the right cheek than was found on the left side.

The zygomatic region of the elongation side is hypoplastic compared with the normal left side. The lip commissure is almost horizontal. The left lip corner seems more laterally placed than the right. The amount of mouth opening is normal but with deviation to the left side, less than in the closed position. Viewed from below, the elongation of the right side, the deviation of the chin and the hypoplasia of the right infraorbital-zygomatic region are more impressive than in the front view. In profile view there is nothing abnormal.

All permanent teeth are present (Fig. 47d–f). The occlusal plane is horizontal. There is a typical crossbite occlusion on the left side. The right molars are in a class III occlusion while on the left side there is a slight class II occlusion. The midline of the lower teeth is shifted to the left by the width of 1 1/2 lower incisors.

Radiographs. The panoramic view in the closed position (Fig. 47g) shows a rather peculiar picture for an

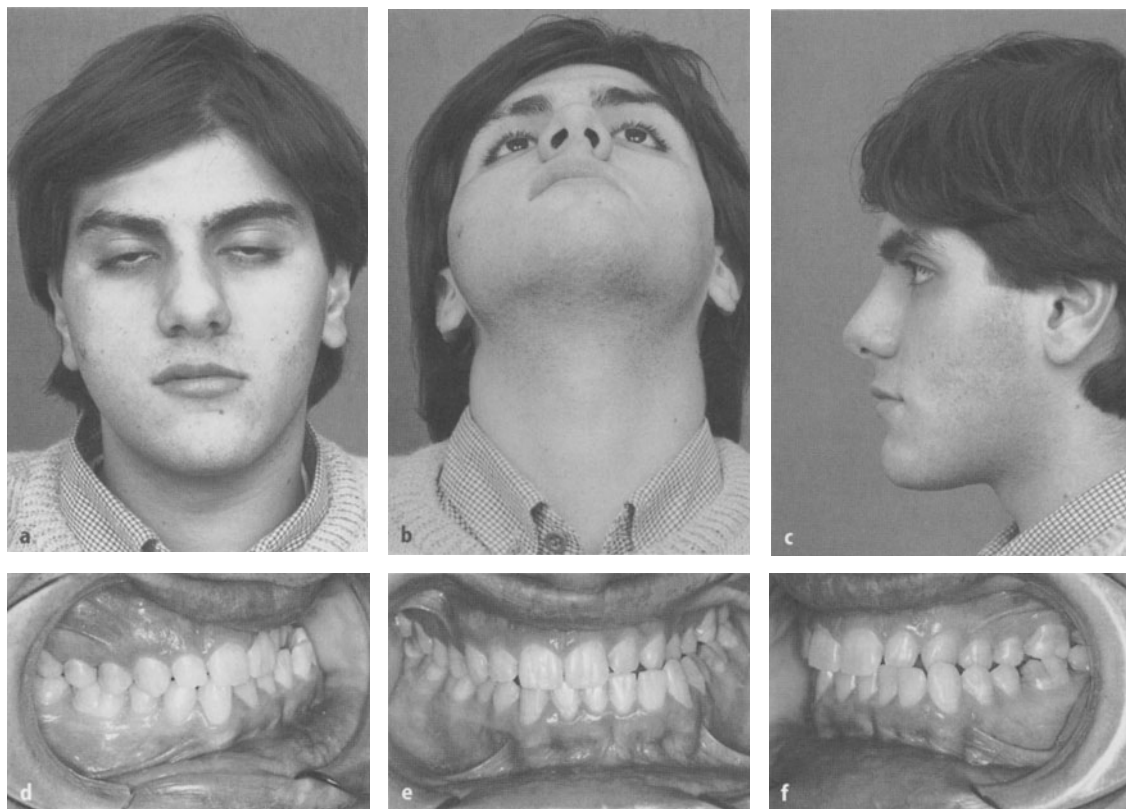


Fig. 47a–f. Unilateral H.E., medium-slender type. **a–c** The patient's appearance typical of an unilateral H.E. before surgery, but with a more voluminous contour on the elongation side. The profile does not show the unilateral elongation. **d–f** Occlusion typical of an unilateral H.E. of the right side

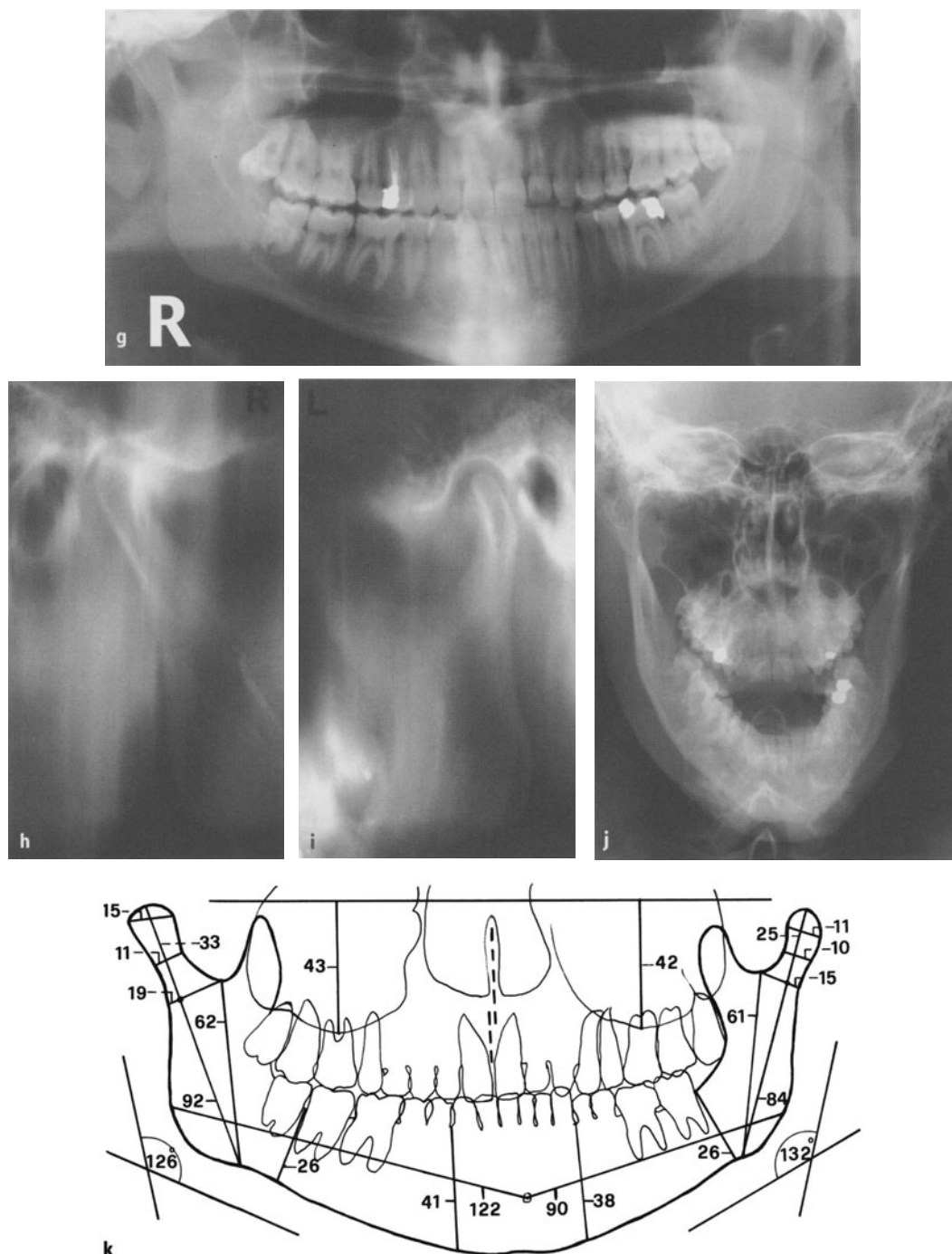


Fig. 47g-k. Unilateral H.E., medium-slender type. **g** The panoramic view shows the pronounced deviation of the chin, but the neck-condyle situation is not very typical of a right side H.E. **h, i** The tomograms of the TMJ including ramus show the clear picture of an unilateral H.E. **j** The mandible in p.a. projection in the open mouth position leaves no doubt about a right side H.E. **k** The tracing of the panoramic radiograph and its measurements prove clearly the diagnosis of an unilateral H.E.

H.E. asymmetry: the right condyle is not clearly definable from the neck. The latter seems to be a part of an elongated condyle. There is a wide open angulation from the articular process to the posterior border of the ascending ramus and there is not, as is usual, an elongation of that posterior border. The right horizontal ramus of the mandible is clearly longer than the left, while the ascending ramus including the condyle seems to have less height than that on the other side. Whether the

left body and ramus are of normal length and height is difficult to say. They seem to be a bit on the short side. The left condyle seems somewhat smaller but not really hypoplastic. Its neck is almost in one line with the posterior border of the ascending ramus. The two angles differ remarkably. The right one is of normal shape while the left is almost absent. The crossbite to the left and the tilting of the right front teeth to the elongated right side could have been overlooked. Both mandibular

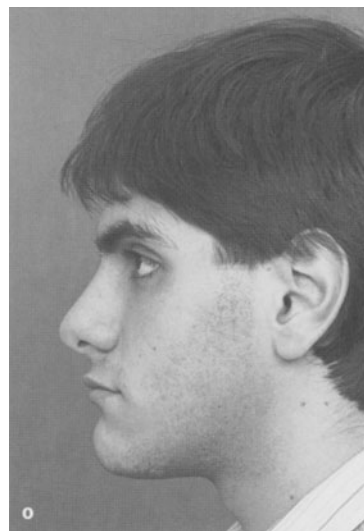
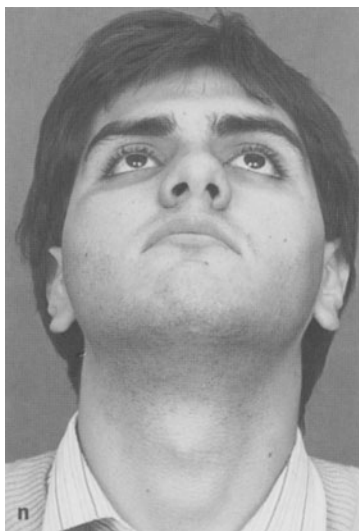
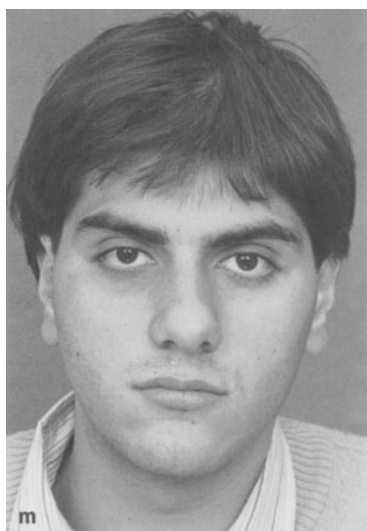
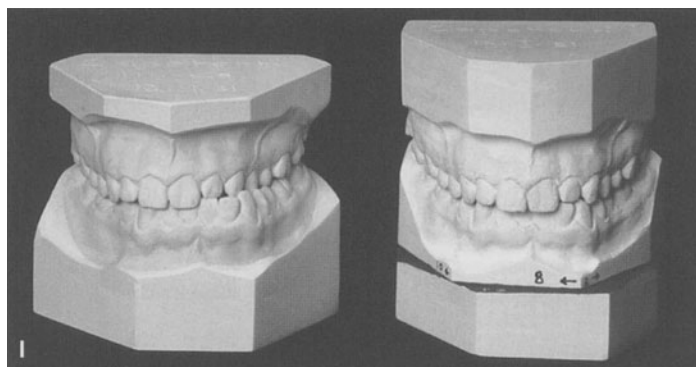


Fig. 471–r. Unilateral H.E., medium-slender type. **l** According to the model operation a very acceptable occlusion could be achieved by a bilateral sagittal splitting procedure on the rami. **m–o** The patient's appearance 1 year after correction: good symmetrization and no change in profile. **p–r** The patient's occlusion after 1 year: there was no orthodontic treatment

horizontal rami are of normal height. The trabeculation structure seems normal on both sides. The position of the mandibular canal is normal.

In the tomograms of the TMJ including ramus (Fig. 47h, i) the two sides differ remarkably. On the left side, the condyle and its neck are rather slim but still within normal limits in shape and size, although rather long. The right condyle also seems larger in size than the left but also within normal limits and there is a clearly elongated neck with a less pronounced angulation of the neck to the posterior border of the ascending ramus. This is different to that observed on the panoramic view.

The mandible in p.a. projection in the open position (Fig. 47j) shows the very pronounced difference in length between right and left sides, with the normally shaped chin well out of the facial midline. In the angle region the right side seems clearly thicker than the left.

The lateral cephalogram discloses that the two horizontal rami seem slightly unequal in height. But the profile line is in no way prognathic.

The measurements of the tracing of the panoramic view (Fig. 47k) make it clear that it is mainly the right articular process and horizontal ramus which are longer than those on the other side, while the length of the trunk on the two sides is almost equal.

Provisional Diagnosis. Hemimandibular elongation of medium slender type on the right side.

Treatment Plan. According to the model operation (Fig. 47l) a good occlusion and facial symmetry will be achievable by a bilateral sagittal splitting procedure on the rami and lyophilized cartilage onlays to the right infraorbital-zygomatic area.

Actual Treatment and Result. The correction of the asymmetry was carried out according to the plan. The required result was achieved without pre- or postoperative orthodontics. There was no negative change in the profile line, but a very slight difference in cheek volume remained (Fig. 47m-r).

Discussion. This case of H.E. was somewhat atypical for several reasons. The elongation of the right condylar neck, as seen in the TMJ tomograms, could not be so clearly diagnosed in the panoramic view. The length of the right condylar neck suggested that this was a slender type of H.E. and yet there was a rather normal mandibular angle on the affected side. There seemed to be some lack of body height on that side as there was an antegonial depression, but measurements of the tracing could not confirm it. So the depression was due to an enlargement of the angle area, probably as a consequence of a medium degree of masseter hypertrophy.

The contralateral side lacked the normal mandibular angle. It was an angle type that a slender H.E. type would have and yet its ramus and body were shorter than in a normal class I case. This raised the question whether or not this case would have been a retromandibulism case if no unilateral H.E. had developed and also whether the surplus of soft tissue volume and contour in the right angle was an unilateral masseter hypertrophy of medium degree or whether there was soft tissue hypoplasia on the left side.

Conclusion. For correct diagnosis of the abnormality of the mandible, a panoramic view alone is not enough. One must, in addition, have tomograms of the TMJ including the ascending ramus. This case also showed that the growth abnormality does not always develop according to the classical form of the abnormality. It also demonstrated the difficulty in diagnosis as we do not know what the position of the patient's mandible would have been if no additional growth regulation abnormality in this type of condylar growth hyperactivity had occurred. It further demonstrated that in some cases the elongation is due mainly to the elongation of the articular process and the horizontal ramus which shifts the mandible over to the opposite side. It also reveals how much existing differences in soft tissue volume between the two sides can influence the clinical picture, making the diagnosis even more difficult. The question might also arise why the angle on the H.E. side was less stretched than that on the normal side. This may be due to the existing masseter hypertrophy.

Unilateral Highly Active H.E., Slender Type, at Puberty (Fig. 48)

Sex and Age. Female, 12 years and 11 months.

Aetiology and History. During the past few months, an already pre-existing slight mandibular asymmetry had increased remarkably. The parents did not know when they first noticed it nor did they remember any possible cause for it. The parents wanted to limit radiographs to a minimum. In order to know whether the hyperactivity was still ongoing, a wait of 6 months was suggested, as the parents would not permit scintigraphy. The girl returned after 5 months because the facial asymmetry had increased very much, the mandible had come forward and the previously existing left crossbite had now turned into an open bite.

Clinical Findings. The almost 13-year-old girl shows an asymmetry of her mandible, with the chin deviated about 8 mm to the left (Fig. 48a). There is a rather pronounced labio-mental fold with the lower lip in a slightly forward position. The facial musculature and inner-

vation are normal. In the profile view, the lower lip is in correct relationship to the upper (Fig. 48b). There is no class III appearance whatsoever. When opening the mouth the chin remains in the midline (Fig. 48c).

The occlusion is badly disturbed with a class III occlusion on the right side and a left crossbite with a class II occlusal relationship (Fig. 48d–f).

Radiographs. In the panoramic view projection (Fig. 48g), the mandibular asymmetry is clearly noticeable with a higher ramus-condyle section and a longer body section on the right side than the left. The right angle is more obtuse compared with the left. The right condyle with its neck is clearly longer than the left. Neither the condyle nor its neck seem enlarged in volume. The left condyle and its neck seem normal in size and configuration. The right mandibular lower border is very close to the apices of the molars. Also on the “normal” left

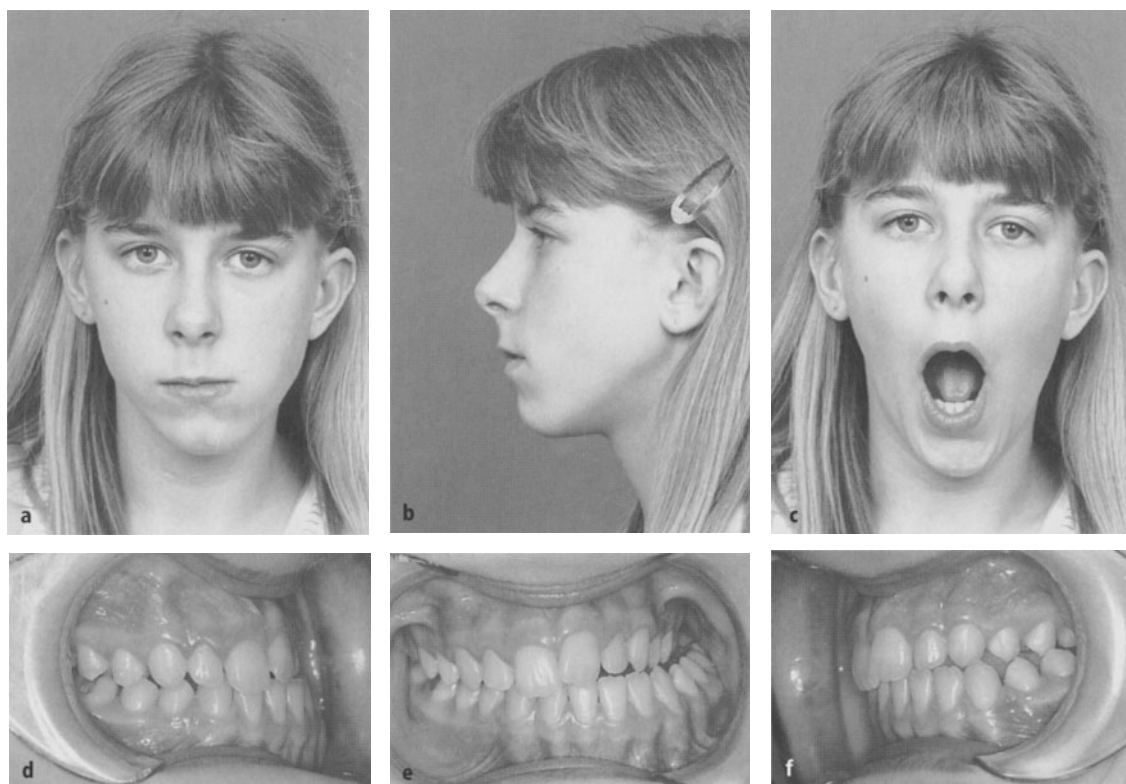


Fig. 48a–f. Unilateral highly active H.E., slender type, at puberty. **a–c** The patient's appearance at 12 years and 11 months from in front and profile and with maximum mouth opening with some deviation of the mandible towards the shorter left side. **d–f** The patient's occlusion on the same day as the facial photographs were taken

side there is a lack of body height compared with normal. The trabecular structure is unremarkable in the entire mandible and the mandibular canal is almost invisible in the horizontal part of the mandible. The prominence of the chin as well as the midline of the lower teeth were shifted to the left by the width of a lower incisor. The second molars are not yet fully erupted.

The tomograms of the TMJ and ramus area (Fig. 48h, i) show a normal situation on the left side. On the right side the condyle is also of normal size but its neck is longer and slightly thicker than that on the left.

The mandible in p.a. projection at maximum opening (Fig. 48j) shows that the upper first incisors are exactly in the facial midline. Some type of orthodontic appli-

ance is worn in the maxilla. There is quite a difference in length of the two ascending rami. The right ramus seems elongated and slim, the left is rotated and therefore seems broader than normal. The chin prominence and the lower teeth are positioned far over to the left side.

The lateral cephalogram disclosed nothing of significance, no signs of any asymmetry or irregularity of the profile.

The wrist radiograph showed that all epiphyseal plates of the fingers and the radius and ulna were still clearly distinguishable.

The tracing of the panoramic radiograph (Fig. 48k) proves that in a H.E., slender type, all sections of the af-



Fig. 48g-j. Unilateral highly active H.E., slender type, at puberty. **g** The panoramic view shows an asymmetrical mandible with a longer right side and deviation of the lower teeth midline to the left by the full width of a lower incisor. **h, i** The TMJ tomograms including the rami and angles disclose that the right articular process is remarkably longer than the left and its condyle is smaller than that on the left side. **j** The mandible p.a. projection at maximum mouth opening also shows clearly the elongation of the right side of the mandible with deviation to the left side

affected hemimandible may be longer than those of the normal side, much longer than one would have expected. The right horizontal ramus is 24 mm longer than that on the normal side. The ascending rami have a difference in height of 10 mm and the articular processes

and the trunks 4 mm. The condylar width is 3 mm less on the elongation side and the width of the necks is equal. The angle on the affected side is very stretched compared with the angle on the normal side. There is an 11° difference.

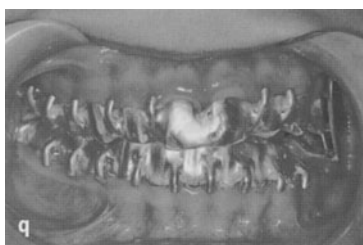
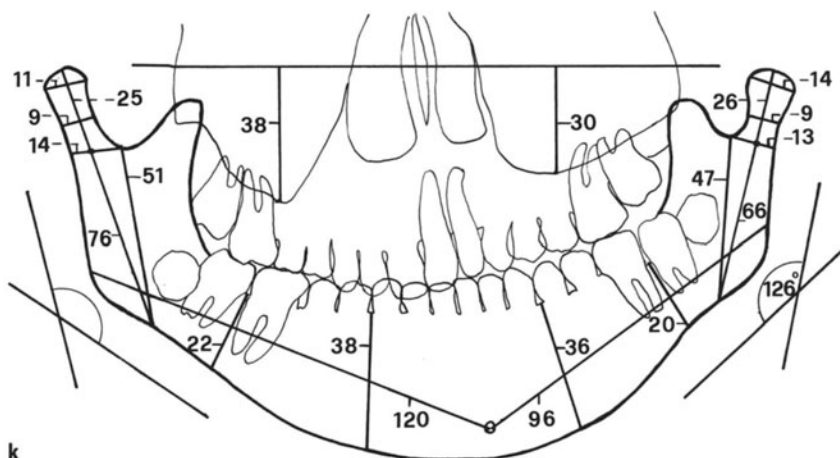


Fig. 48k–q. Unilateral highly active H.E., slender type, at puberty. **k** The measurements of the tracing of the panoramic radiograph disclose all the typical signs we expect in a H.E., slender type. **l, m** The patient's appearance 5 months later: in the front view it looks like an "asymmetrical prognathism", but there is nothing of this visible in the profile view. **n–p** Within 5 months the condylar hyperactivity had shifted the occlusion further to the left by the width of half an incisor and has produced an open bite. **q** The cemented cast cap splints with training flanges on the non-operated side control mandibular movement without deviation

Clinical Findings 5 Months later. Five months later (Fig. 48l,m), the chin asymmetry has turned into a facial asymmetry with protrusion of the lower lip, the mandible and chin in an asymmetric, nearly antemandibulism (class III) appearance. However, in profile view there is no sign of antemandibulism. At maximum mouth opening the mandible and chin now clearly deviate towards the elongated right side.

The occlusion on the right side indicates that the mandible has come further forward, and on the left side there is an open bite in addition to the crossbite (Fig. 48n-p). *Scintigraphy* was not permitted by the parents.

Provisional Diagnosis. Highly hyperactive unilateral H.E. on the right side at the age of 12 years and 11 months.

Treatment Plan. Intracapsular high condylectomy on the right side via a retrotragal approach. Postoperative insertion of cast cap splints with training flanges for 6–8 weeks to prevent mandibular deviation when opening and closing. After their removal, postsurgical orthodontics.

Actual Treatment. The high condylectomy was carried out at the age of 13 years and 4 months. The postoperative course was uneventful. The cap splints were cemented 9 days after surgery (Fig. 48q) and were removed 6 weeks later. The girl then had normal mouth opening without deviation and an acceptable occlusion. Additional postsurgical orthodontic treatment was still indicated. Eight months after surgery there was absolute facial symmetry (Fig. 48r-t). The further development of the mandible was normal and 3 years later the good result achieved surgically is still unchanged.

Histology. Macroscopically, the resected specimen was of fairly normal shape and size, its medio-lateral width being about 15 mm and its antero-posterior length about 11 mm. Microscopically, condylar bone along the articular surface was covered by hypertrophic growth cartilage of normal thickness, a cell-rich proliferative layer and an articular layer that was damaged at one end of the specimen (Fig. 48u). Almost exactly at the centre of the articular surface, the articular layer was markedly thickened in a cone-shaped way, thus displacing the proliferative and hypertrophic layers downwards, both of which were unchanged in thickness (see Fig. 79c). While the shape of this localized protrusion was reminiscent of a so-called vascular canal, such as is observed frequently in condyles from infants, the connective tissue of the articular layer did not disclose any blood vessels. Active endochondral ossification at the lower border

of the hypertrophic cartilage was revealed by chondroclasts and seams of osteoblasts lining the primary marrow spaces as well as by subchondral cartilage rests contained in the trabeculae of the spongiosa. Notably, these cartilage rests were confined to a zone of about 2.5–3 mm in thickness, as is also observed in control specimens. Unlike in the latter, however, the marrow spaces of the spongiosa were large, while trabeculae were thin and showed a clear orientation, sometimes largely parallel (see Fig. 79c).

Diagnosis. Actively growing condyle exhibiting a circumscribed thickening of the articular tissue of unknown significance and a conspicuous spongiosa characterized by few slender, almost parallel trabeculae.

Comment: Whereas no signs of enhanced condylar growth could be detected, the observed architecture of trabeculae in the subchondral spongiosa appears typical of H.E., irrespective of whether they are classified as slender or non-slender. In contrast to these features of the spongiosa, a cone-shaped thickening of the articular layer could not be observed in any of the other H.E. cases and, therefore, can hardly be considered a distinguishing characteristic.

Discussion. Hyperactivity of the L condylar growth regulator can all of sudden become so severe that even in H.E. cases an early high condylectomy becomes mandatory. The forward push of the affected side can turn a primarily class II case into a class III on the affected side while the contralateral side remains in its original class II position. For both psychological as well as skeletal and occlusal reasons, in such a case one cannot wait until mandibular growth has ceased. Since postoperatively mandibular development seems to return to normal in spite of the missing upper half of one condyle, there arises the question whether or not one should, in all cases of H.E., suggest a very early high condylectomy, as we definitely do for the H.H. and the hybrid cases.

Histologically, no signs of enhanced growth could be found in the resected part of the condyle in spite of the fact that there was a gross increase in the H.E.-anomaly until the operation. And yet the architecture of the trabeculae observed in the subchondral spongiosa was typical of H.E. As a clinician I would expect clear signs of growth hyperactivity in the condyle in such a case, irrespective of lack of proof by scintigraphy.

Conclusion. In cases of very severe hyperactivity of the condylar L growth regulator, high condylectomy is essential independent of the age of the patient. In these cases there is a very clear histological differentiation between normal mandibular growth or even a H.H. or a hybrid form.

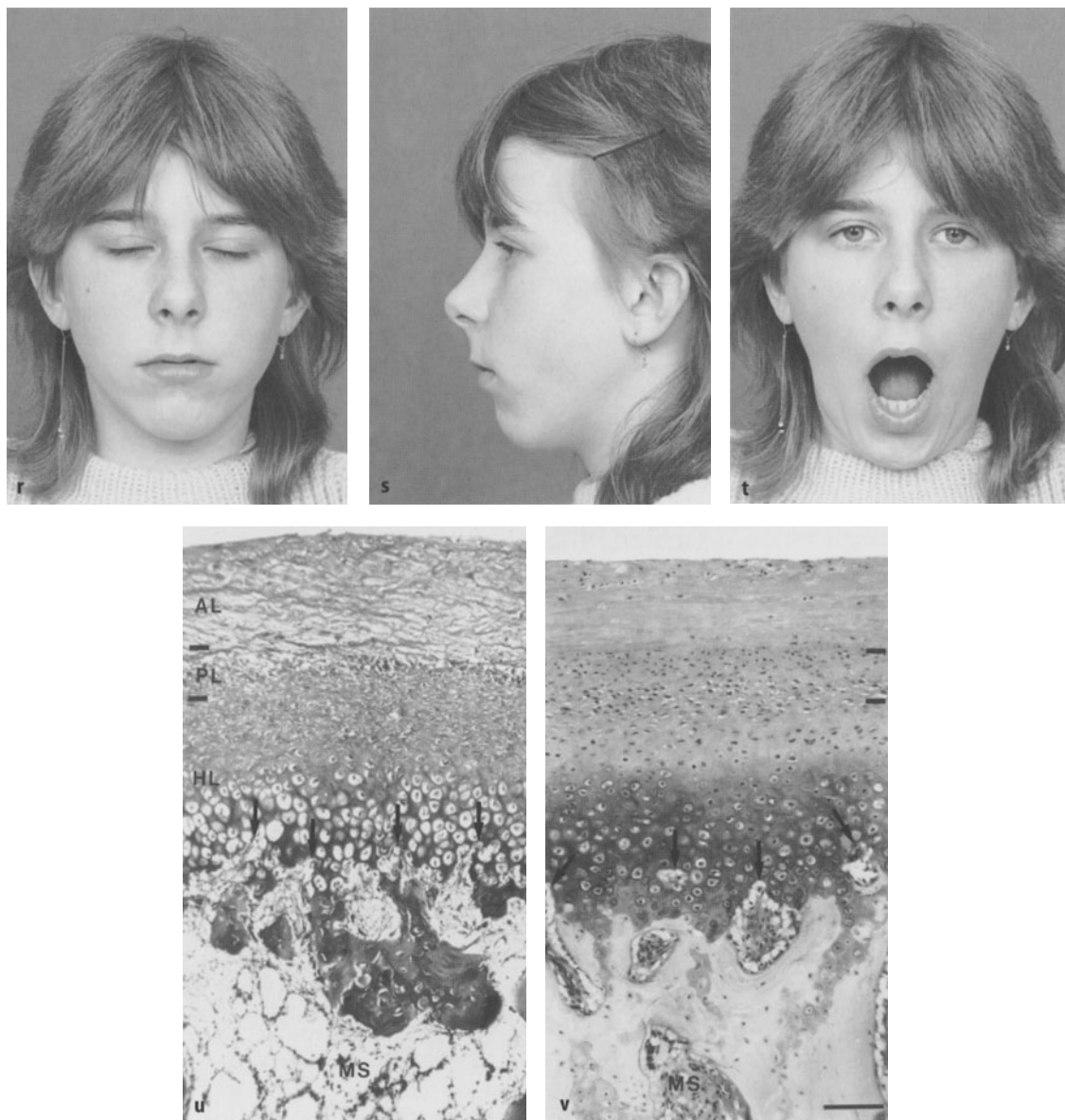


Fig. 48r–v. Unilateral highly active H.E., slender type, at puberty. **r–t** Eight months after condylectomy, the patient's face is symmetrical and the mouth opening without any deviation. **u–v** Microscopic appearance of the articular tissue of the excised condyle **u** in comparison with that seen in a normal specimen from a female of about 14.5 years of age **v**. Note the normal thickness of the hypertrophic layer **HL**, the extensive resorption lacunae forming the lower border of the cartilage (*arrows*) and the large marrow spaces **MS** of the primary spongiosa in the affected condyle **u** (**AL** articular layer, **PL** proliferative layer, **u** paraffin section, **v** plastic section, toluidine blue, original magnification $\times 130$, bar = 100 μm)

Unilateral H.E., Slender Type, Associated with Rotation of the Maxilla (Fig. 49)

Sex and Age. Female, 18 years of age.

Aetiology and History. The patient was a forceps delivery. She had no idea of the cause of her facial asymmetry. Already before puberty, at about the age of 10, her chin had moved slightly towards the left side. At the age of 12–13 years the asymmetry became much worse. There had been occlusal problems as long as she could remember. But with the chin moving to the left side the occlusal situation became worse. Her dentist had tried to correct the asymmetry of her occlusion with orthodontic treatment but had not been successful. For the past year there had been no further change. Therefore, she was referred for surgery.

Clinical Findings. Looking at the patient from the front (Fig. 49a), the asymmetry of the mandible is very striking. The chin prominence is over to the left by at least 15 mm. There is a slightly oblique lip commissure with the left corner a bit higher and more lateral than the right. In profile view the patient's face was that of a

mandibular long face, with a retruded chin (Fig. 49b). The facial soft tissues and muscle innervations were normal. The mouth opening was within the normal range, with some correction of the asymmetry.

Teeth 16, 36 and 46 are missing. When closing the teeth, there is a very severe crossbite on the left side. The midline of the lower teeth is shifted to the left by the width of $2\frac{1}{2}$ lower incisors in relation to the midline of the upper teeth (Fig. 49c–e). There is slight lingual inclination of all her mandibular teeth with minor tilting of the front teeth towards the longer right side. An orthodontic lingual bar is fixed to the lower teeth.

Radiographs. The panoramic view (Fig. 49f) discloses that the severe crossbite asymmetry is only partly due to the mandible. The maxilla seems rotated to the right side by the width of one upper first incisor and the mandible is rotated to the left by the width of one lower incisor. The shape of the mandible is asymmetrical with

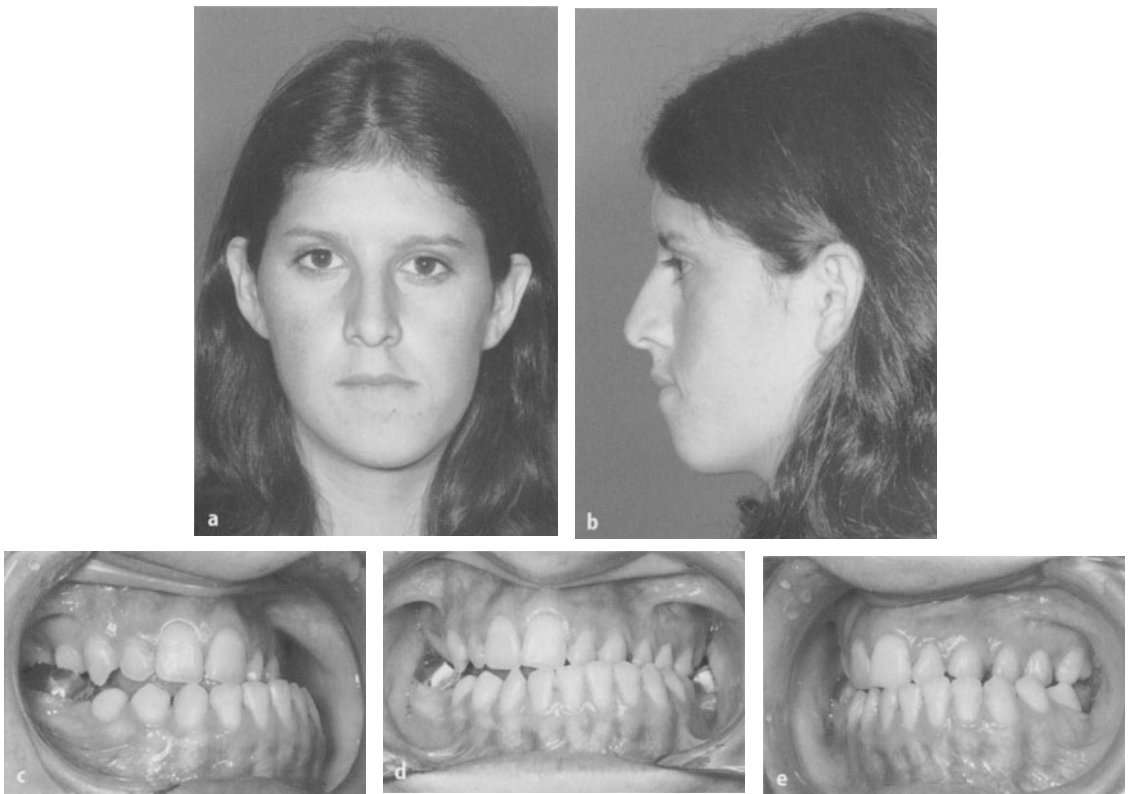


Fig. 49a–e. Unilateral H.E., slender type, associated with rotation of the maxilla. **a, b** Front and profile views of the patient show severe deviation of the chin, elongation of the right side of the mandible, slight class III profile type and retrognathia. **c–e** The occlusion shows a rotational discrepancy between the upper and lower midlines of the teeth by the width of $2\frac{1}{2}$ lower incisors

its left half within normal limits in form and length including the condyle and its neck and the angle. The right half is elongated. Its condyle is slim and the condylar neck is rather thin and straight and longer than nor-

mal. Although a mandibular angle clearly exists, the mandible seems stretched in that angle area, making the whole right half of the mandible slightly longer than the left. All other radiographs were missing. Nevertheless it

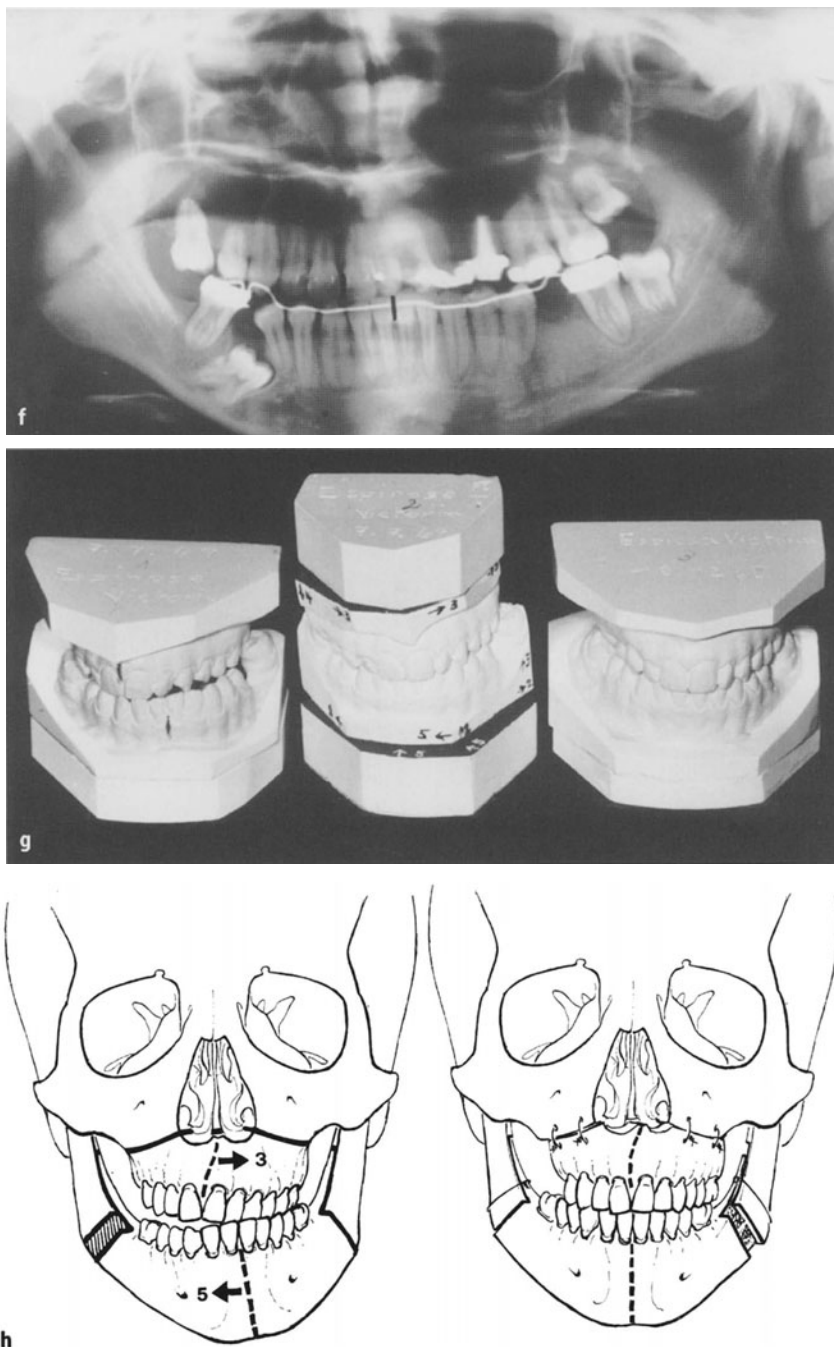


Fig. 49f-h. Unilateral H.E., slender type, associated with rotation of the maxilla. **f** The panoramic view alone discloses the anatomical background of this anomaly. **g** The model operation with a bimaxillary rotation achieves a very satisfactory occlusal situation. **h** Diagrammatic illustration of the planned correction with a rotation of the maxilla to the left and of the mandible to the right side

seems worthwhile to present this case for teaching purposes.

Provisional Diagnosis. Unilateral hemimandibular elongation, slender type, on the right side and slight retromaxillism with rotation of the maxilla to the right in addition.

Treatment Plan. The model operation (Fig. 49g) discloses that a very acceptable occlusion and correction of the crossbite can be achieved as soon as both jaws are completely mobilized. For this reason it was planned to perform a Le Fort I type mobilization of the maxilla for its anterior repositioning and rotation to the midline, and a bilateral sagittal splitting procedure on both rami for correction of the mandibular asymmetry and the crossbite (Fig. 49h). Pre- and postsurgical orthodontics were suggested, but the patient refused this as she thought the occlusion and the facial symmetry she will acquire with surgery is all that she wanted.

Actual Treatment and Result. The surgery was carried out according to the plan, followed by 6 weeks intermaxillary fixation. A very good correction of the facial asymmetry was achieved (Fig. 49i,j) and also an ac-

ceptable occlusion, without presurgical orthodontics (Fig. 49k-m). An additional advancement of the chin prominence and reduction of the chin height was suggested. But the patient refused.

Discussion and Conclusion. Although a rather severe mandibular asymmetry due to an unilateral H.E. can shift the midline of the lower teeth quite a bit over to the other side, it is rather doubtful if it could be by the width of $2\frac{1}{2}$ lower incisors. In such a case, one will have to determine whether there is an additional shortness of the contralateral side of the mandible due to a hemimandibular hypoplasia, or whether it is due to a rotation of the maxilla. Although the latter is rather rare, the possibility exists, as in this case, without any definite knowledge of its aetiology. It may be that the forceps delivery had displaced the maxilla slightly. The proper treatment plan does presuppose a correct diagnosis. In this case, the extent of the asymmetry might have suggested the existence of very extensive hyperactivity of the L growth regulator and that a wrong diagnosis might have indicated an early condylectomy. That would not have prevented the necessity for correcting the existing rotation and slight retrodisplacement of the maxilla.

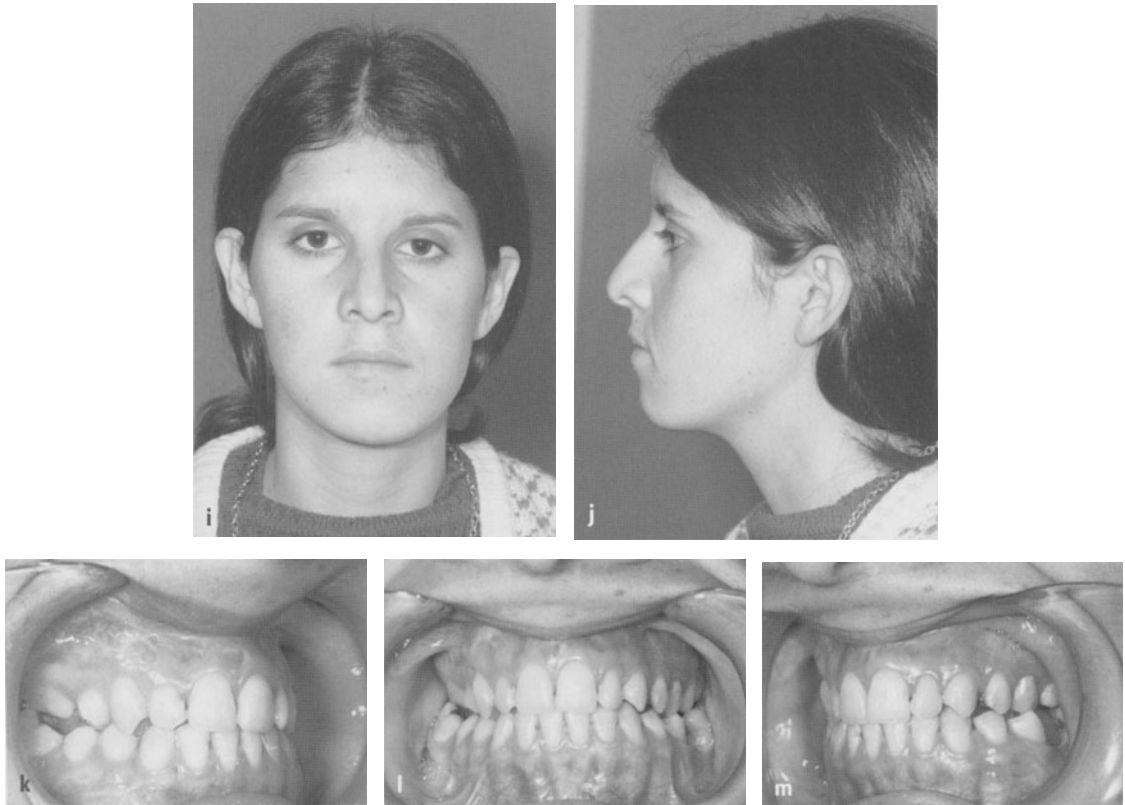


Fig. 49i-m. Unilateral H.E., slender type, associated with rotation of the maxilla. **i, j** The patient's front and profile views 1 1/2 years after surgery. **k-m** The occlusal situation achieved by surgery alone

Unilateral H.E., Slender Type, in a Case of Secondary Cleft Lip and Palate Deformity (Fig. 50)

Sex and Age. Female, 17 years of age.

Aetiology and History. The patient came from a foreign country. She was born with a left-sided complete cleft lip and palate deformity. The lip and palate closure was performed very early. The parents did not know what kind of technique was used. They, as well as the young lady, were of the opinion that the whole deformity was due to the cleft lip and palate. They sought normalization of her facial, occlusal and speech deformities. The patient had had orthodontic treatment for the past 2 years.

Clinical Findings. A very typical secondary cleft lip and palate deformity is present (Fig. 50a–c), with the middle third of the face and the nose very flat, and the mandible in an antemandibulism position. The infraorbital rims and paranasal areas are very flat. The nose is large and shows the typical secondary deformity of an unilateral cleft closure, and its ridge and tip seem deviated to the right side. The upper lip is short and scarred in the cleft region. The mandible seems normal in size and configuration. It has free movement and no difficulties in opening. The chin seems to be about in the midline of the face and does not deviate on mouth opening. There is the usual lack of facial height due to micro- and retro-maxillism (maxillary short face).

The occlusion (Fig. 50d–g) shows the usual disaster following early radical primary cleft closure. There is a circular open bite situation with occlusal contact only of

the last molars. The lower teeth are tilted lingually and the front teeth tilted additionally to the right side. The upper teeth are crowded as they do not have enough space in the small retrodisplaced upper jaw. The midline of the lower front teeth is to the left by the width of 1 1/2 lower incisors compared with the upper midline.

As the lower incisor midline seems to be to the left side by quite an amount, the facial symmetry had to be evaluated more accurately, using the standard technique on the enlarged photograph (Fig. 50h). It proved the following: The nasal bridge including the tip of the nose is exactly in the midline of the face. The chin prominence is deviated severely to the left. The left zygoma is clearly hypoplastic and the left eye seems larger than the right. The right ascending ramus region is flatter than the left and the left angle region is more voluminous than the right.

Radiographs. The *orthopantomogram* (Fig. 50i) discloses the very typical mandibular abnormality of an unilateral H.E. of the slender type. Both condyles seem normal in size and shape, but the right has a longer and narrower neck than the left. The height of the ascending rami including condyles seems almost equal on both sides. However, the right angle is clearly stretched although still rounded. On both sides there is a mild antegenonial concavity. There is a fine trabecular structure in the whole mandible including the condyles.



Fig. 50a–c. Unilateral H.E., slender type, in a case of secondary cleft lip and palate deformity. **a–c** The patient's front and profile views showing the profile type of a severe antemandibulism with a typical unilateral cleft nose deformity

The measurement of the tracing (Fig. 50j) reveals that the right horizontal ramus measures 5 mm more in length than the left but its vertical height is much less in the molar region than the normal left body. The difference measures 9 mm. The measurement also discloses that the H.E. on the right side is less severe than anticipated, according to the asymmetry evaluation. The larger angle on the left side seems to compensate for the excess length of the right horizontal ramus to a certain degree.

The tomograms of the TMJ (Fig. k, l) show a normal left condyle with a normal neck. The right condyle

seems smaller and has a long slim neck of about the same diameter as the condyle. Regrettably, the ascending rami are not shown on these radiographs.

The mandible in *p.a.* projection in maximum opening also disclosed a length difference between the two ascending rami, though very moderate. The symphysis was clearly out of the facial midline to the left, but not very much.

The lateral cephalometric view (Fig. 50m) shows the clinically impressive mid-facial hypoplasia with lack of vertical height and sagittal length. It also shows that the

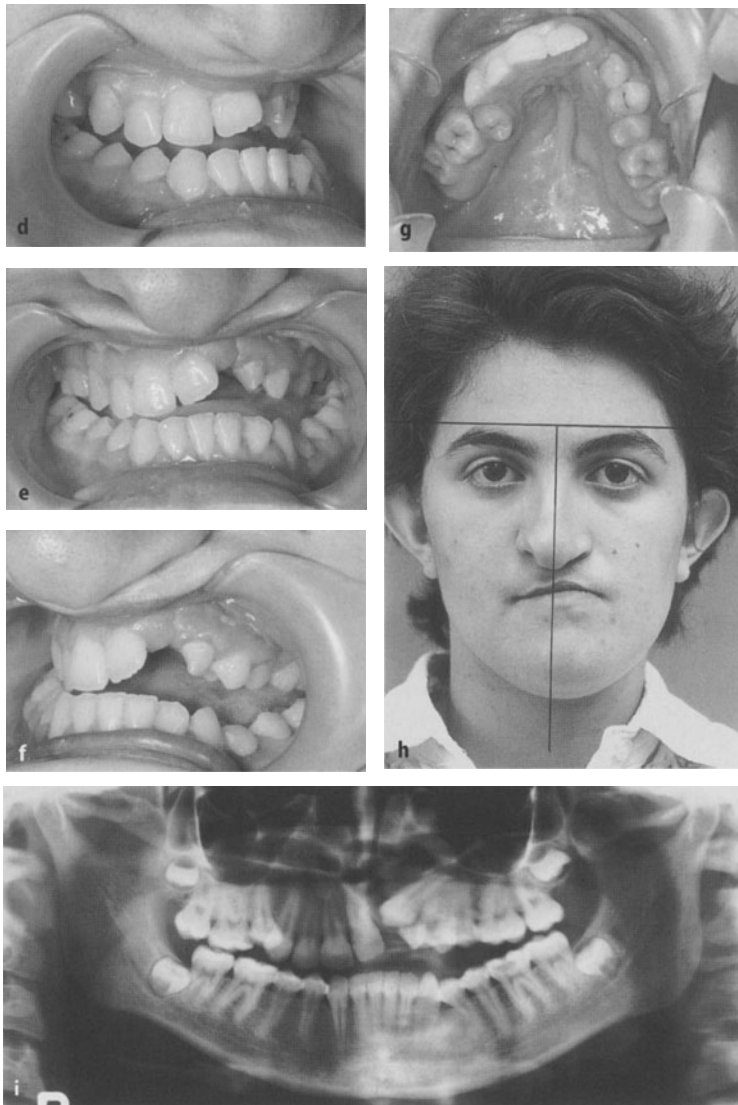


Fig. 50d–i. Unilateral H.E., slender type, in a case of secondary cleft lip and palate deformity. **d–g** Typical occlusion of severe retro- and micromaxillism in a unilateral cleft case and collapse of the upper arch. **h** Asymmetry evaluation of the patient's front view: contrary to the clinical diagnosis there is no deviation of the nasal bridge but of the chin prominence and hypoplasia of the left zygoma region. **i** The panoramic view promotes the diagnosis of severe H.E., slender type, on the right side

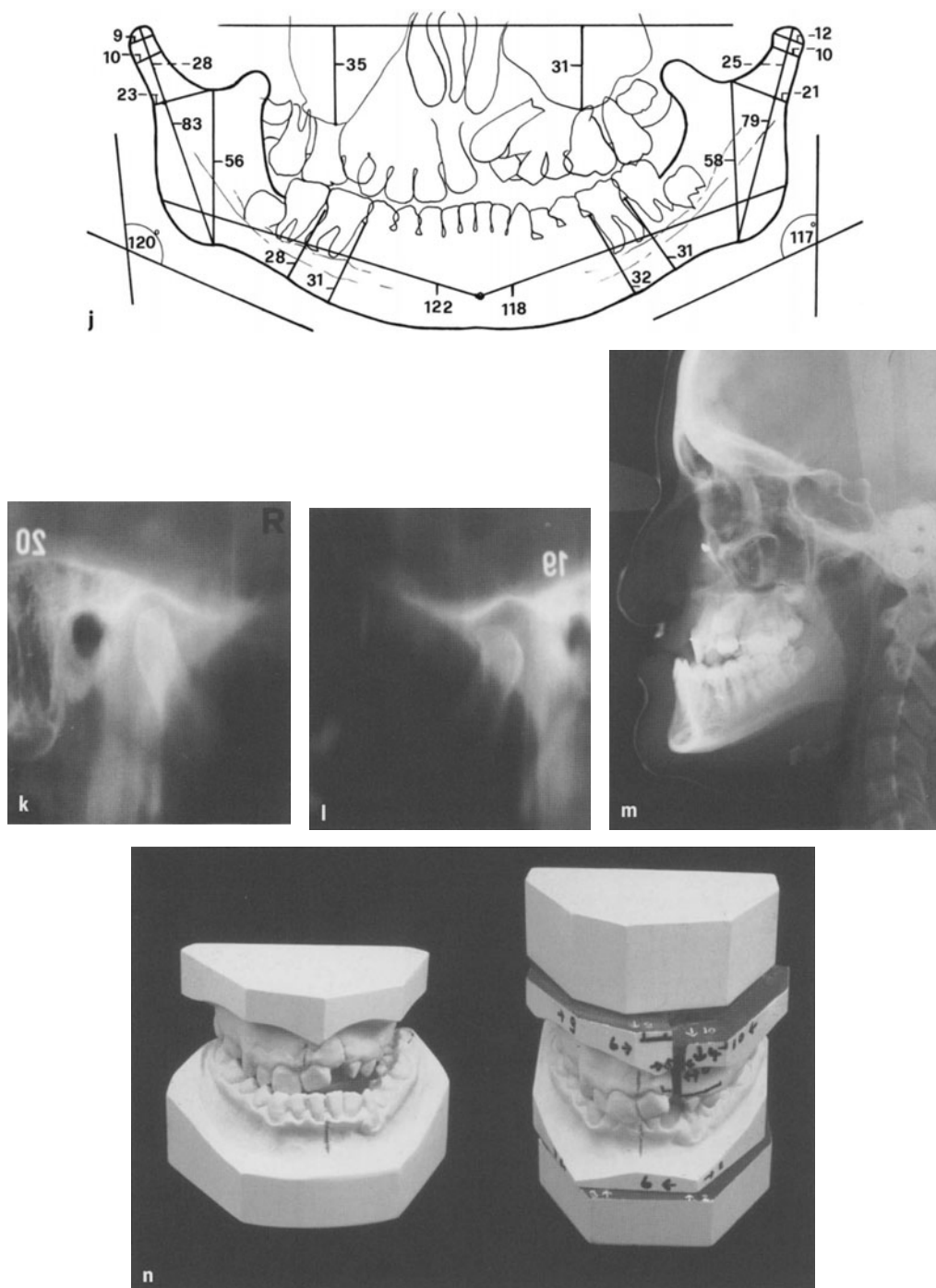


Fig. 50j-n. Unilateral H.E., slender type, in a case of secondary cleft lip and palate deformity. **j** The tracing of the panoramic view discloses that the differences in length between right and left sides are less than anticipated. **k, l** In the TMJ tomograms there is the typical condyle-neck situation of a H.E. anomaly recognizable on the right side, although of a rather mild degree. **m** The lateral cephalogram discloses that the patient's antemandibulism appearance is mainly due to the retromaxillism. **n** The model operation gives information about the necessary amount of movement of the mandible and of the two unequal halves of the maxilla

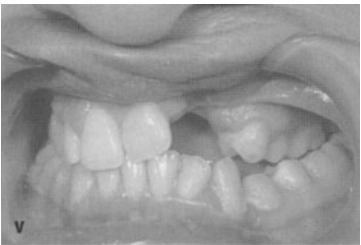
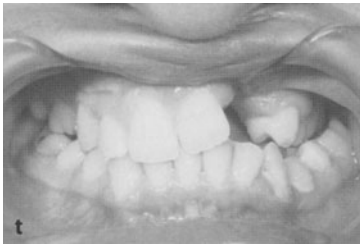
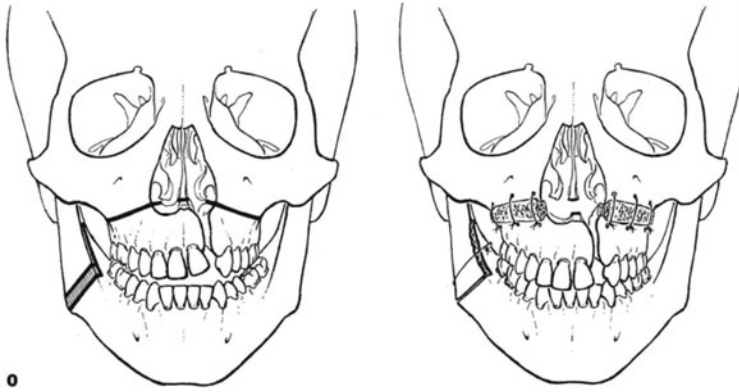


Fig. 50o-w. Unilateral H.E., slender type, in a case of secondary cleft lip and palate deformity. **o** Diagrammatic illustration of the type of osteotomies planned for correction of this severe facial deformity. **p** The lateral cephalogram taken 1 1/2 years after the correction of the skeletal anomaly proves that the plan was correct: advancement of the two halves of the maxilla only with rotation of the mandible to the elongated side, but without any repositioning of the mandible, producing a straight antepile.

q-s The patient's face and nose after correction of the skeletal deformities and additional lip elongation, using the Le Mesurier technique, and simultaneous correction of the cleft nose, 2 years after correction of the skeletal deformity. **t-w** The occlusion and the maxillary arch 3 1/2 years after correction of the skeletal deformity and closure of the reopened cleft: collapse of the small segment had to be expected without the necessary postsurgical orthodontics

mandible is of normal length and not like an antemandibulism type.

Provisional Diagnosis. Classical slender type of unilateral H.E. in a case with mid-facial hypoplasia due to retro- and micromaxillism related to a unilateral secondary cleft deformity.

Treatment Plan. As the patient came from outside Switzerland it was agreed that she should have the necessary surgical correction without presurgical orthodontics, as her orthodontist could not improve the situation within 2 years time, for some reason or another. Orthodontic treatment to be carried out postoperatively as well as possible. By correcting the occlusal deformity on a model operation (Fig. 50n) the necessary osteotomies and amount of repositioning of the skeletal parts was determined. In accordance with the model operation, the surgical correction is to be performed in 3 stages.

Stage 1 (Fig. 50o): Anterior repositioning of the maxilla in two segments with reopening of the palate to widen the maxillary arch. Simultaneously, a sagittal splitting procedure of the ramus on the elongation side only, will correct the mandibular abnormality and guarantee that it will not be shortened; direct bone wiring and 6 weeks intermaxillary fixation will be used to secure bony union in the desired position. For increase in vertical height of the maxillary complex and for securing bony union and preventing a relapse, cancellous bone grafts will be wedged in the Le Fort I type osteotomies in the canine fossa region and in the retromaxillary space.

Stage 2: Six months later, the reopened palate will be closed by the use of local tissues and pieces of a thinned half rib graft, and a pharyngoplasty will be performed simultaneously.

Stage 3: Some months later, the lip will be reoperated upon to elongate it, and the cleft nose deformity will be corrected.

Actual Treatment. The surgery was carried out as planned in three stages. The sagittal discrepancy between the severely retrodisplaced maxilla and the pseudo-antemandibulism was corrected mainly by advancing the two halves of the maxilla. With the additional corrective surgery on the lip and nose deformities a straight anteproof resulted (Fig. 50q-s). The occlusal situation did not turn out as planned. Primarily, all maxillary segments had been repositioned into their correct places and occluded properly with the teeth of the mandible. Three years later no satisfactory orthodontic treatment had been started and the lesser maxillary segment had collapsed again.

Discussion and Conclusion. An H.E. may easily be overlooked in the presence of an additional asymmetric deformity of the upper jaw. It becomes very obvious only

when the diagnosis is made on the basis of the evaluation principle: *“When you are not sure whether a facial asymmetry exists or not, place the patient’s head in the upright position, then draw a horizontal line above the eyebrows and to it an imaginary line perpendicular to the middle of the forehead. It can then be seen clearly whether a facial asymmetry exists or not and what is on which side of the face and what the differences are in the two sides of the face”* (Principle No. 15). For proper planning and a successful result, the following principle of Hans Pichler, my “professional grandfather”, is very important: *“The facial skeleton is the framework that determines the shape of the face. Therefore, in facial reconstruction, first the bone then the soft tissues”* (Principle No. 3). Clear diagnosis, adding one sign to the other, and good standardized radiographs, led to the correct diagnosis in this case and also to the correct treatment plan and result.

The H.E. slender type, in its signs and radiological appearance, is easy to diagnose. In this case I did not feel it necessary to perform a sagittal splitting procedure on the rami bilaterally. The decision whether or not one should operate on both rami is very easy. If the H.E. side is done first, one will then find out whether or not the correct position of the mandible can be achieved with that one side osteotomy only. The rotation in the contralateral joint has never caused any TMJ problems in these unilateral osteotomy cases. I doubt whether or not in such a case, an early high condylectomy would be advisable and also be the correct therapy. It seems, based on the results of high condylectomy in the very actively growing cases of H.H. and the hybrid forms that one might consider performing early high condylectomy also, in all H.E. cases; but preferably not in cases where an anomaly of the maxilla has also to be corrected.

A remark on the collapse of the laterally placed small fragment of the maxilla in cleft cases: when the two halves of the maxilla have to be separated and the cleft reopened for repositioning the maxillary segments into the planned position, I see no way of reclosing it simultaneously using local tissue and bone. We must wait until bony union in the new position prevents further collapse. For that, at least 4–6 months retention of the segments in the new position must be achieved by the use of a splint or an orthodontic appliance. It is difficult enough to understand why some patients do not want or accept it, except for financial reasons. If nothing is secured the collapse of the lesser segment will be certain. In cases where one tooth only is missing in the cleft alveolus I prefer to close that gap by advancing the lesser segment so far forward that it contacts the greater segment (H. Obwegeser 1988). In these cases the chance of a collapse will be much less, as we will have bony union in the canine fossa as well as in the alveolar arch. However, also in such a case, the new position of the lesser segment must be secured by some type of orthodontic appliance.

Questionable Unilateral H.E., Slender Type, and Contralateral Hemimandibular Hypoplasia (Fig. 51)

Sex and Age. Female, 12 years and 9 months.

Aetiology and History. At the age of 10 years, an asymmetry of the chin attracted the school dentist's attention. That asymmetry increased evidently during the following 2½ years. At the age of 12 years and 9 months the girl was referred for evaluation and clarification of

her mandibular asymmetry and for treatment suggestions, because of the rapid increase in her mandibular and occlusal asymmetry from age 10 years and 1 month to 12 years and 3 months (Fig. 51a, b).

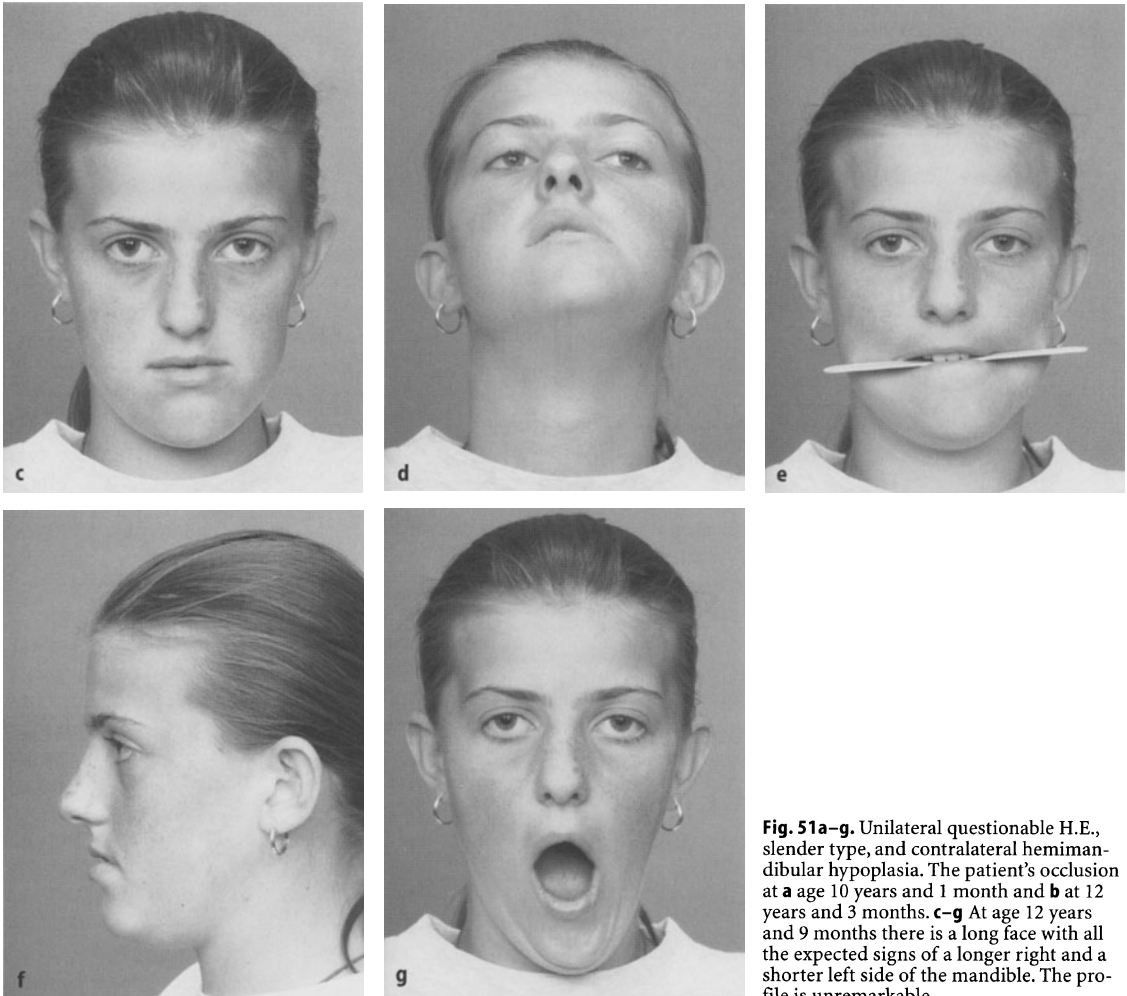
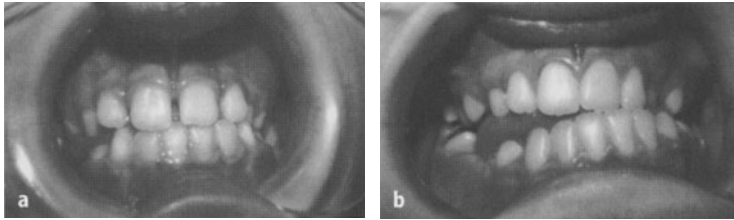


Fig. 51a–g. Unilateral questionable H.E., slender type, and contralateral hemimandibular hypoplasia. The patient's occlusion at **a** age 10 years and 1 month and **b** at 12 years and 3 months. **c–g** At age 12 years and 9 months there is a long face with all the expected signs of a longer right and a shorter left side of the mandible. The profile is unremarkable

Clinical Findings. The girl has a slim longish face (Fig. 51c). The chin prominence is shifted well to the left (Fig. 51d). The lip commissure is almost horizontal with its left corner more lateral than the right.

The right cheek has a normal rounded contour, the left is remarkably flatter with the usual infra-zygomatic concavity. The right mandible seems remarkably longer than the left. There is quite an oblique occlusal plane, ascending to the left side (Fig. 51e). In the profile view there is nothing noteworthy (Fig. 51f). The mouth opening is within the normal range, but with deviation to the left side (Fig. 51g).

When closing the teeth, the severe mandibular asymmetry of the girl's face which is seen from in front is also clearly seen in the occlusion (Fig. 51h-j). But the occlusal picture suggests that there is not only a shift of the mandible to the left but in addition that the occlusal plane on the left side is a little higher than on the right side. However, this is mainly due to the fact that the left canine and incisors are much higher than those on the right, due to a tilting of the left mandible as well as the teeth. In addition there is lingual tilting of the lower incisors and in spite of that there is a slight class III overbite in the anterior region, with the midline of the lower teeth shifted to the left by nearly the width of a lower first incisor. Although the bicuspid and the upper second molars are not fully erupted there is a slight open bite on the right side.

Radiographs. The panoramic view in the closed position (Fig. 51k) reveals the skeletal background to the girl's mandibular asymmetry: There is a clear difference between left and right sides. The right condyle seems fairly small, with a slim elongated neck. The ascending ramus on the right side makes a bit of an obtuse angle with the body, but 130° is within the range of normal. Compared with the normal, there is perhaps a bit of lack of vertical height of the right body in the molar region, so much so that the space between the inferior rim and the apices of the molar roots is just enough to accommodate the mandibular canal. The midline of the upper teeth seems to be almost exactly in the facial midline while the lower midline is shifted to the left by nearly the width of a lower incisor. The lower incisors and the canines are clearly tilted to the longer right side. The distance from the right angle to the symphysis seems longer than normal as well as longer than that on the left side. But it is difficult to say whether the left horizontal ramus of the mandible is shorter than normal. The left ascending ramus is rather short with a very short and very thin condylar neck and a mini-condyle. The left angle also nearly shows a normal contour (124°) and there is a bit of an antegonial notch. There is a normal dentition in the upper jaw but rather large interdental spaces in the lower. Tooth 35 is missing.

There does not seem to be any difference in height between the two sides of the maxilla.

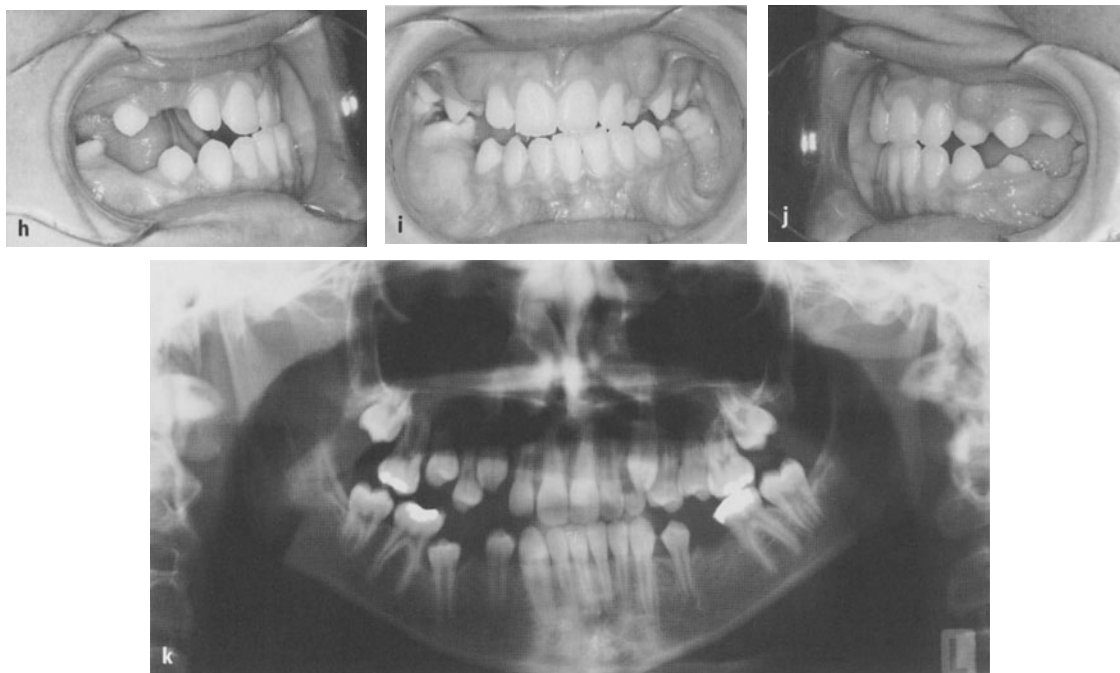


Fig. 51h-k. Unilateral questionable H.E., slender type, and contralateral hemimandibular hypoplasia. **h-j** The occlusion also reflects the longer right and shorter left sides. **k** The panoramic view discloses the long right and short left sides, the latter with a very small condyle and neck

The TMJ–ramus tomograms (Fig. 51l, m) are of rather poor quality. Nevertheless the left mini-condyle with its short and thin neck is clearly visible and so is the rather small right condyle and its neck.

The mandible in *p.a. projection* at maximum mouth opening (Fig. 51n) shows clearly an elongated right mandible in comparison with a rather short and rotated left hemimandible.

Hand-wrist radiograph: The epiphyses of the fingers were almost completely closed, but not so those of the ulna and radius.

The measurement of the tracing (Fig. 51o) of the panoramic view reveals that the right ascending ramus is 14 mm longer than the left, with an 8 mm difference in the length of the two articular processes and 5 mm between the two trunks. The right horizontal ramus is 4 mm longer than the left. There is no noticeable differ-

ence in the height of the two sides of the maxilla. The left angle measures 6° less than the right which at 130° is within the normal range.

Scintigraphy: The report from the Institute of Nuclear Medicine of the Kantonsspital Aarau states that there were no indications of circumscribed osseous disturbances of transformation in the skull region and in particular in the area of the TMJs.

Provisional Diagnosis. Questionable, almost inactive H.E., slender type on the right, and slight hemimandibular hypoplasia on the left side with a left sclerosed mini-condyle and condylar neck.

Treatment Plan. In order to avoid the development of psychological problems, the asymmetry should be corrected as soon as possible in spite of the patient's age of

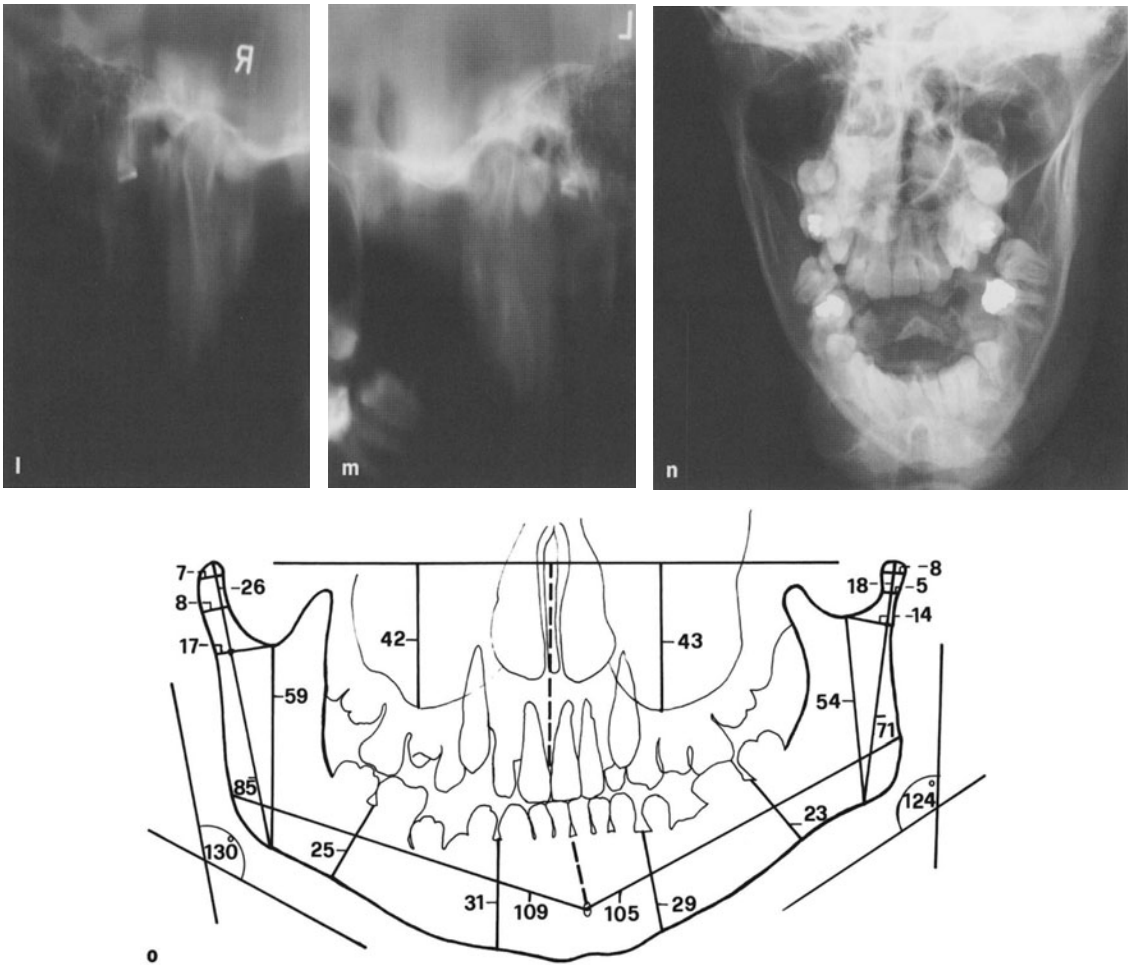


Fig. 51l–o. Unilateral questionable H.E., slender type, and contralateral hemimandibular hypoplasia. **l, m** The TMJ–ramus tomograms disclose a clearly too small left articular process, while the right is within the range of normal. **n** The *p.a.* projection of the mandible in the open mouth position confirms the clearly elongated right hemimandible compared with the twisted left side. **o** The measurements of the panoramic view tracing indicate that all sections of the right hemimandible are longer than the corresponding parts of the left

only 12 years and 9 months. A high intracapsular resection of the right condyle should be performed in order to prevent further elongation of the right hemimandible. In addition a sagittal splitting of the right ramus may be necessary for correcting this severe asymmetry (Fig. 51p). The maxillary long face and possibly later, minor remaining asymmetry of the mandible should be corrected after the eruption of all teeth and after growth has ceased.

Actual Treatment. After the high, intracapsular right side condylectomy the asymmetry due to excess length of that side was not corrected adequately. In addition, a sagittal splitting of the right ramus was performed in order to be able to shorten the right side more and to

correct the mandibular asymmetry sufficiently. Screw fixation of the fragments secured their position and allowed the patient to open her mouth immediately after the surgery. The asymmetry of the mandible was almost completely corrected (Fig. 51q), except for a certain amount of flatness in the area of the left horizontal and ascending rami and a slight remaining deviation of the chin (Fig. 51r, s).

Histology. No abnormal findings in the resected condyle, in particular no hyperactivity, only activity consistent with the patient's age.

Discussion. Because of the rapid increase in the asymmetry of the mandible at the age of 9–12 years, the slim

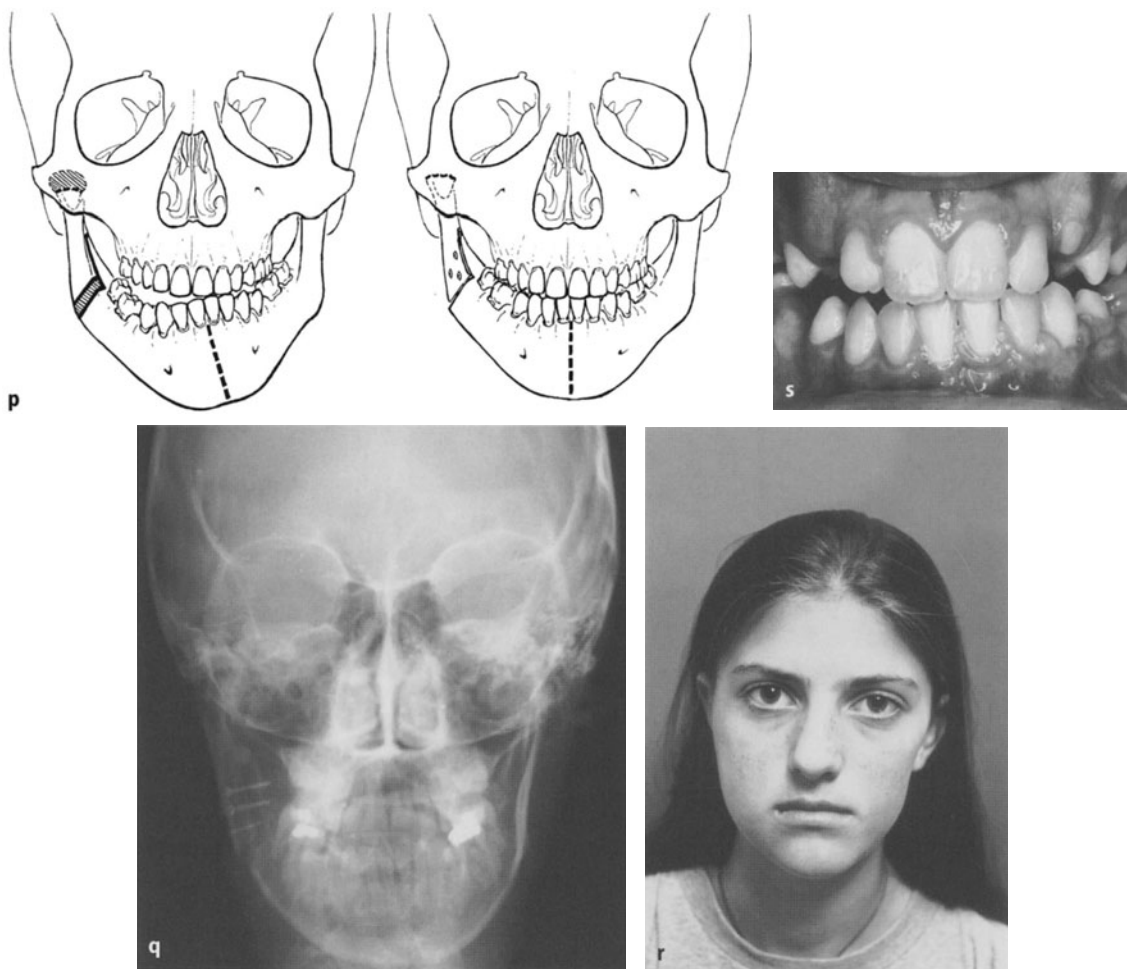


Fig. 51p–s. Unilateral questionable H.E., slender type, and contralateral hemimandibular hypoplasia. **p** Schematic illustration of the planned surgery: intracapsular high condylectomy on the right side and sagittal splitting of the ramus on the same side. **q** The p.a. cephalogram, taken 3 weeks after surgery, shows that the asymmetry is extensively corrected. **r, s** The patient's front view and occlusion 6 months after surgery

and rather elongated right condylar neck, the rather flat right angle, and the reduced body height in the molar region, one is tempted to make a diagnosis of H.E., slender type, on the right side. However, the very hypoplastic and sclerotic condyle and neck on the left side suggests that a hemimandibular hypoplasia on the left side may be the cause of the asymmetry. But the left ramus and angle lack the typical shape of a longstanding condylar hypoplasia. The ascending ramus is slightly short, but the angle is within normal and the very typical antegonial notch is not present. If that condyle had been damaged before or soon after birth, the ramus should be even less high and the maxilla should not have grown down equally as on the elongation side. Therefore, the severe damage to the condyle must have occurred relatively recently. This is also proven by the fact that there is a class I molar relationship on the same side. So the asymmetry is mainly due to the possible H.E. of the right side.

Another point has to be taken into consideration. That is the cause of the patient's prognathic crossbite in the anterior region. A pure unilateral H.E. normally shifts the mandible over to the opposite side only, and not into a class III anterior relationship. Therefore, the question arises whether this girl is a primary antemandibulism case which has lapsed into a class I occlusion on the left side due to its hypoactive condyle and re-

mained on the right side as an antemandibulism with a class III occlusion. The slightly higher condylar cell activity of the right side is, of course, no proof that it is a hyperactivity case since some normal growth activity may still be present and since the left side is definitely hypoactive. The histology of the resected condyle also seems to support this.

So, we are not able to diagnose for certain the aetiology of this case's mandibular asymmetry. An early condylectomy was the only way to guarantee that there will be no further overgrowth of the right side and, together with a simultaneous simple unilateral sagittal split procedure, it corrected the asymmetry to such an extent that the young girl can develop without any psychological complex due to facial deformity. The very excellent orthodontist who had referred this case will be able to prepare the occlusion properly for the surgery proposed after cessation of growth. I wish to thank Dr. H.U. Schwitzer from Solothurn for this very interesting case.

Conclusion. There are cases which present such complex possible aetiological factors for their "simple" mandibular asymmetry that we are unable to verify their real background even with scintigraphy. In such a case additional histology alone can elucidate the problem. Nevertheless, the type and timing of the treatment must be clear in our minds.

Unilateral H.E., Non-Slender Type, Commencing in Early Childhood (Fig. 52)

Sex and Age. Female, 19 years and 5 months.

Aetiology and History. The parents reported that at the age of 4–5 years the chin had moved forwards but that the asymmetry was first noticed at the age of 16 years. There had been no further increase for the past year. The parents and the patient could not remember any possible reason why it should have started. The patient did not complain of any TMJ pain. No pertinent family history.

Clinical Findings. Looking at the patient from in front (Fig. 52a), a clear mandibular asymmetry is recognizable, with elongation of the left side. The prominence of the chin is over to the right by about 1 cm. The left cheek is slightly less prominent than the right. The lip commissure is slightly oblique, descending towards the longer side. The facial musculature and nerves are normal. In profile the young lady has a slight asymmetrical antemandibulism appearance (Fig. 52b).

Mouth opening is unhindered but with deviation to the right side. When closing the teeth the mandible deviates step-like to the right side.

In centric occlusion (Fig. 52c–e) there is a crossbite on the right side and the midline of the lower front teeth is shifted to the right by the full width of a lower incisor. On the left side there is a severe class III occlusion. On the right side there is also a mild class III relationship in the molar region. But a normal overbite remains.

Radiographs. The panoramic view (Fig. 52f) shows the classic picture of a H.E., non-slender type. The right side shows a normal condyle but with a rather short neck (perhaps due to inadequate radiological technique) and a normal ascending ramus and body of the mandible. The right angle is also normal. The chin midline is shifted to the right by 7 mm and so is the midline of the lower teeth. The left mandible is clearly elongated compared with the normal right side. Its angle is stretched but still a bit rounded. The left condyle is normal in size and shape. Its neck is rather broad and longer than the right but within the normal range. The left body lacks mandibular height in the molar region, compared with the normal, but that is also true of the right

body. The bony structure shows a very fine trabeculation and the mandibular canal has little space between the apices of the teeth and the lower border.

The measurements of the tracing (Fig. 52g) of the panoramic view are somehow disappointing, as the difference in length of the various sections of the two sides of the mandible are less than one would expect. The main difference is found in the ascending rami and in

the angles while the length of the horizontal rami differs by 3 mm only.

TMJ tomograms including the ramus (Fig. 52h, i) clearly show that the panoramic view is not distorted. The length of the ascending rami measurements coincide with the figures obtained from the panoramic view. This is also true for the height of the horizontal rami in the molar regions. The measurement of the angles dif-

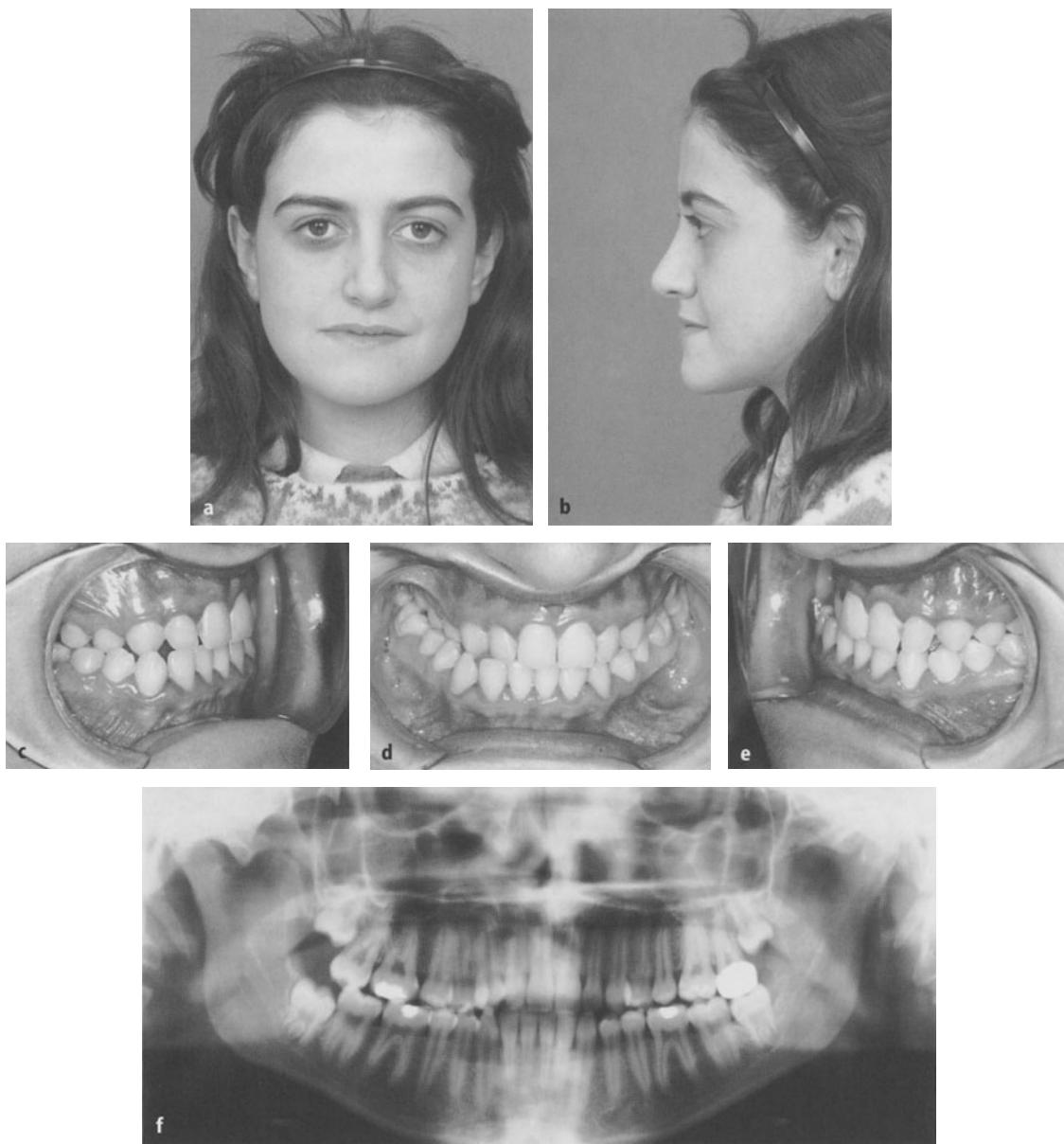


Fig. 52a–f. Unilateral H.E., non-slender type, commencing in early childhood. **a, b** Typical asymmetry of the mandible of the H.E. type, but with a slight antemandibulism influence noticeable in both the front and profile views. **c–e** The occlusion demonstrates the existence of one of the main signs of a H.E. anomaly: the crossbite. **f** The panoramic view discloses the elongation of the left hemimandible compared with the right, but not much else

fers also by the same number of degrees. Only the condyles and their necks could be evaluated better. The right condyle is within the normal range in shape and size. Its neck is rather short. On the left side the condyle is also of normal size and shape but its neck widens remarkably toward the semilunar notch and it is clearly elongated compared with that on the right side. The length of the left ascending ramus is increased compared with the normal right side and the angle is rounded and stretched. There is a very fine trabecular structure on both sides.

The *mandibular p.a. projection* in the open mouth position (Fig. 52j) discloses the elongation of the left mandible very clearly and also its deviation across the facial midline to the other side.

The *lateral cephalogram* shows an almost edge-to-edge relationship of the front teeth, but nothing else worth mentioning.

The *hand radiograph* shows that all symphyseal plates were fully closed.

Provisional Diagnosis. Unilateral H.E., non-slender type.

Treatment Plan. According to the model surgery an almost perfect occlusion should be achievable, without any additional orthodontic treatment, by repositioning the mandible after a bilateral sagittal splitting procedure on the rami.

Actual Treatment. The surgery was carried out according to the plan, using our usual technique, followed by 6 weeks intermaxillary fixation. The result in appearance, profile and occlusion was as expected (Fig. 52k-o). No pre- or postoperative orthodontics was used.

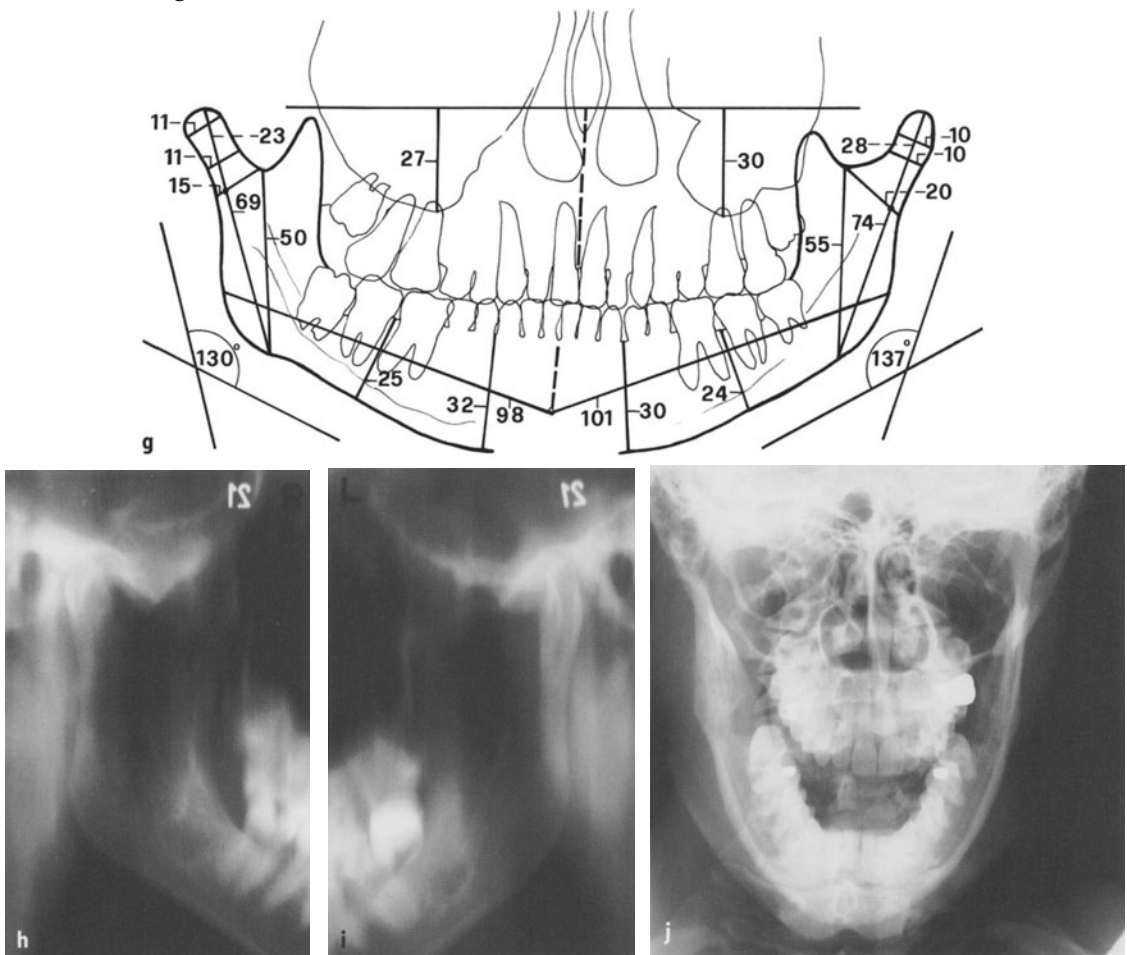


Fig. 52g-j. Unilateral H.E., non-slender type, commencing in early childhood. **g** The measurements of the tracing of this panoramic view disclose that the main difference between right and left lies in the ascending rami and the stretching of the left angle. **h, i** The TMJ-ramus tomograms prove that the left ascending ramus is clearly longer than the right, the trunk as well as the articular process. **j** The mandible in p.a. projection in the open mouth position does not leave any doubt that the left hemimandible is clearly longer than the right

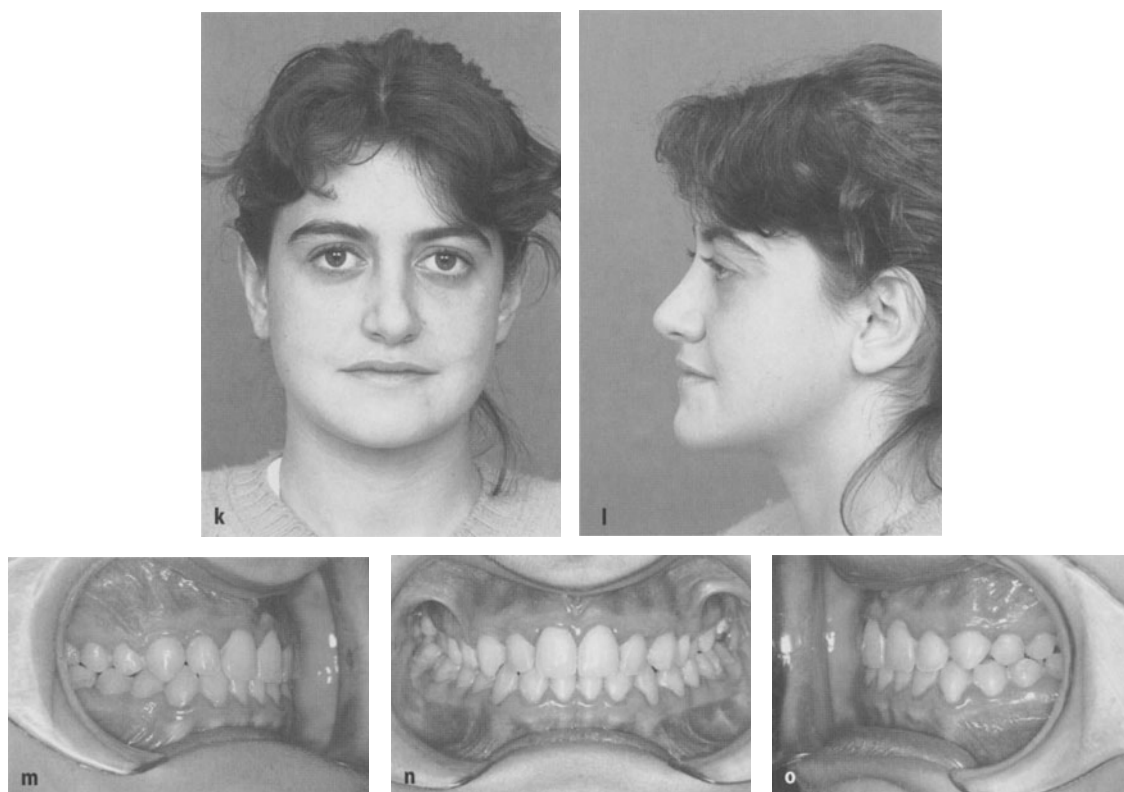


Fig. 52k-o. Unilateral H.E., non-slender type, commencing in early childhood. **k-o** Front and profile appearances and occlusion 1 year after correction

Discussion. Just as there exists a clear slender type of H.E. there also exists a clear non-slender type of H.E. The latter has almost normal condyles compared with the rather small ones of the slender type. The non-slender type has a rather thick condylar neck which is often, but not always, elongated but with a rather broad base. Also the angle seems not as stretched as in a real slender type. Often it seems to have an almost normal angle, although it is always slightly stretched but rounded and not as slim as in the slender type. Whether this non-slender type is due to hyperactivity of the condylar L-regulator only or whether there is an additional minor influence of the M-factor, I do not know. For me it just seems like that.

The history of the case was rather interesting. The parents noticed that the girl's chin moved forward at the age of 4–5 years while the asymmetry started when she was 16. That raises the question whether or not there was first an antemandibulism and only later the asymmetry developed. Without a lateral cephalogram of that age, that question cannot be answered.

Conclusion. Besides the clear slender type of H.E. there also exists a non-slender type of H.E., producing the same clinical picture with crossbite and chin deviation but a somewhat different radiographic appearance.

Unilateral H.E., Still Active, Slender/Non-Slender Type (Fig. 53)

Sex and Age. Male, 15 years and 9 months.

Aetiology and History. The patient and his parents did not know of any possible cause for the mandibular asymmetry. The chin had started to move towards the right side at about the age of 9 years. As it had increased gradually, an orthodontist was consulted. Because of some crowding of the maxillary teeth he had extracted each first upper bicuspid, started orthodontic treatment and referred the patient to my department for consultation.

Clinical Findings. Looking at the patient from in front (Fig. 53a), the mandibular asymmetry is obvious, with the chin displaced to the right side by about 10 mm. The face has the typical appearance what is often called “unilateral prognathism”. The left side of the face is flat and directed straight towards the chin, without the usual cheek prominence. The right side seems over-contoured, in particular in the angle region. The lip com-

missure is slightly oblique, tilting inferiorly on the longer left side. There is a clear difference in facial height between left and right sides, measured from the eyes to the corners of the mouth. Looking at the patient from the side, there is an unobtrusive vertical profile (Fig. 53b). Mouth opening is unaffected, but with clear deviation of the chin to the right side (Fig. 53c).

The occlusal situation looks rather peculiar (Fig. 53d–f). Because of the crowding of the upper teeth and partially of the lower ones also, there is no real occlusion. But the occlusal plane is horizontal. The midline of the lower teeth is shifted to the right by almost $1\frac{1}{2}$ lower incisors width, and there is an open bite, in a crossbite position, on the right side.

Radiographs. The panoramic view (Fig. 53g) shows an almost symmetrical mandible of very similar configuration on both sides, within the normal range. But the

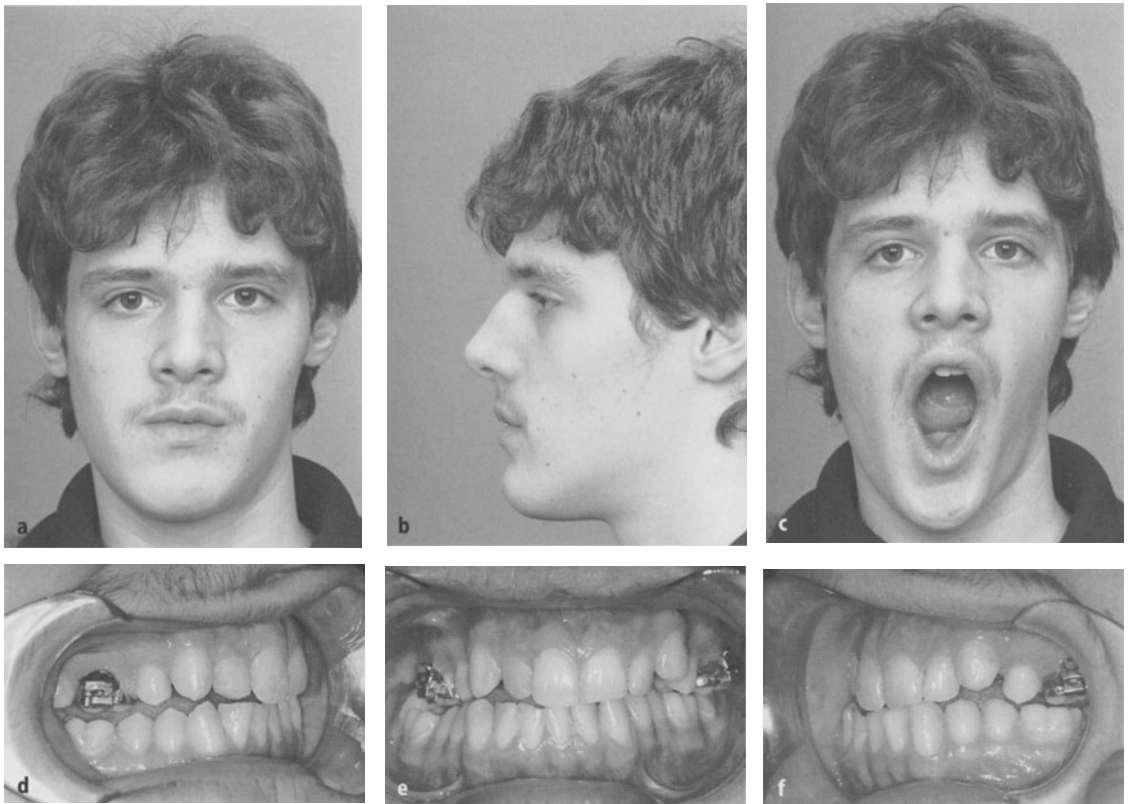


Fig. 53a–f. Unilateral H.E., slender-non-slender type, still active. **a** The patient's front view shows all the expected details of a unilateral H.E., even with an asymmetrical antemandibulism appearance. **b** The profile view corrects that impression. **c** At maximum mouth opening there is a clear deviation to the shorter right side. **d–f** The occlusion shows not only a shift to the right side with clear deviation of the lower midline, but also an open bite on the side of the shorter mandible

left body seems longer than the right. Both condyles are rather large and their necks inconspicuous, except that the left neck seems a bit longer. Although the height of the rami including the condyles looks similar, there is a difference of 4 mm in favour of the left side, measured on this radiograph. Also, the left body measures 17 mm longer than the right. The height of the mandibular bodies is within normal limits, but there is still a difference of 3 mm in the second molar region, shallower on the elongated side. The right angle measures 128° and the left 122° . This means that in this case the angle on the elongated side is not larger. The most prominent part of the chin is in the region of the right second upper incisor, 14 mm out of the facial midline. But the Pg-point as well as the midline of the lower incisors is shifted over to the right side by the width of a first lower incisor only, compared with the midline of the upper

teeth. The upper teeth midline was shifted out of the facial midline (nasal septum) to the left, by the width of one-half an upper incisor. The first upper bicuspid is missing. An orthodontic appliance is fixed to some of the upper teeth. The maxilla seems horizontal.

The *TMJ-ramus tomograms* (Fig. 53h,i) show a normal right condyle with a rather short neck. The left condyle is a bit smaller and its neck is clearly longer than the one on the other side. The difference in ascending ramus height is clearly recognizable. The structure is unremarkable.

The *mandible in p.a. projection* in the maximum opening position (Fig. 53j) permits the diagnosis of a clear elongation of the left ascending ramus and body compared with the normal right side. The right ascending ramus is slightly curved towards the condyle, compensating for the lateral pushing of the right mandible

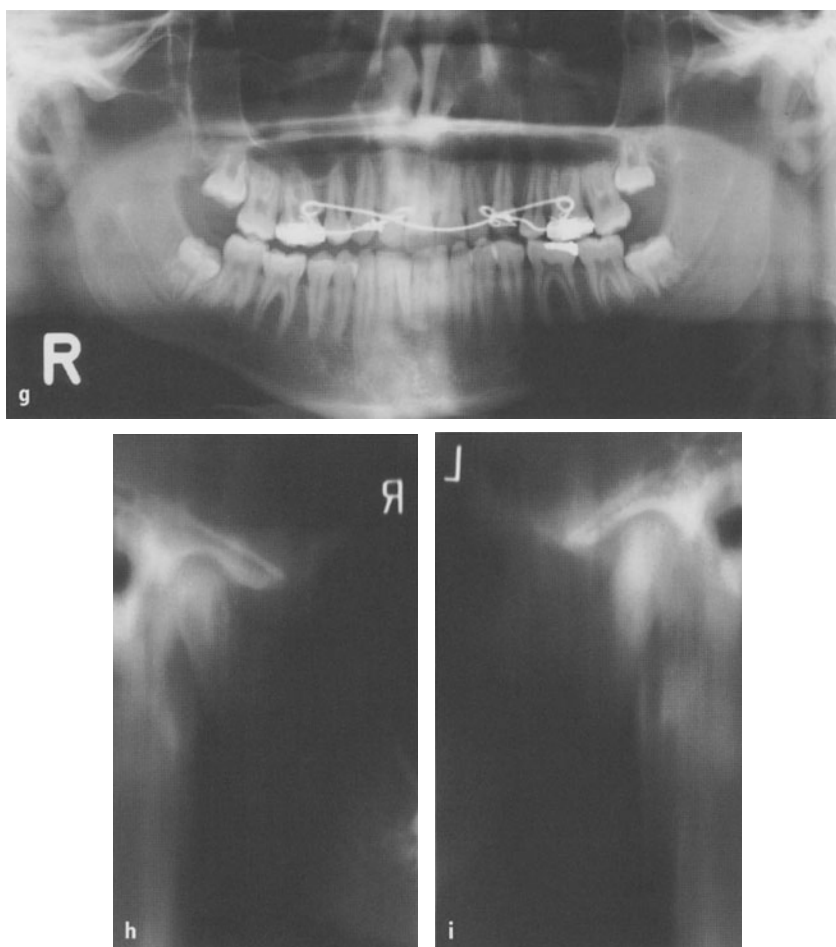


Fig. 53g-i. Unilateral H.E., slender-non-slender type, still active. **g** The panoramic view shows a picture rather typical of a H.E. non-slender type, of the left hemimandible. **h, i** The TMJ-ramus radiographs disclose the difference in the articular processes and ascending rami more clearly than the panoramic view

by the elongated left one. Its structure shows rather coarse trabeculation compared with the fine trabeculation of the left ramus. That ramus appears very straight and clearly longer than the right. In this open position the mandible is shifted well over to the right side. This projection also proves that the midline of the upper incisors is to the left by nearly half a central incisor width.

The lateral cephalogram (Fig. 53k) discloses an almost edge-to-edge anterior teeth relationship, with a maxilla of normal size and position (normomaxillism), but there is not the slightest prognathic appearance.

Wrist radiography revealed that the epiphyseal cartilaginous plates were not far from disappearing completely.

The tracing of the panoramic radiograph (Fig. 53l) permits better comparison of the different sections of the two sides of the mandible.

Scintigraphy of the skeleton with 500 MBq Tc-99m-DPD at the boy's age of 15 years and 4 months showed clearly a concentration in the left condyle, suggesting the existence of condylar hyperactivity, according to the written report. Regrettably, the actual pictures could not be found. For that reason no percentage hyperactivity compared with the normal side can be given and also no RU amount.

Provisional Diagnosis. Unilateral H:E. of a slender/non-slender type with cellular hyperactivity in the left condyle.

Treatment Plan. Immediate intracapsular high condylectomy.

After completion of the presurgical orthodontics and when mandibular growth has ceased, a bilateral sagittal splitting procedure on the rami and an additional symmetrization of the chin prominence should give a normal occlusion and external appearance.

Actual Treatment. High intracapsular condylectomy on the left side was performed. No postoperative appliance therapy was used. The final correction of the anomaly was postponed until the result of presurgical orthodontics will permit it.

Histology. With a medio-lateral diameter of about 15 mm (the antero-posterior width could not be assessed), the removed condyle was apparently normal in size and did not appear deformed. Microscopically, the bone against the articular surface was covered by hypertrophic growth cartilage of normal thickness, a proliferative layer of medium cell density, and an articular layer showing some damage due to surgery along about half of the surface (Fig. 53m). Prominent marrow spaces in contact with the cartilage and containing chondroclasts as well as cartilage rests embedded in the subchondral trabeculae as far down as the resection border indicated active endochondral ossification. Notably, however, condylectomy in this case had been carried out at a distance of only about 3 mm from the zone of erosion. As cartilage rests in control condyles can be ob-

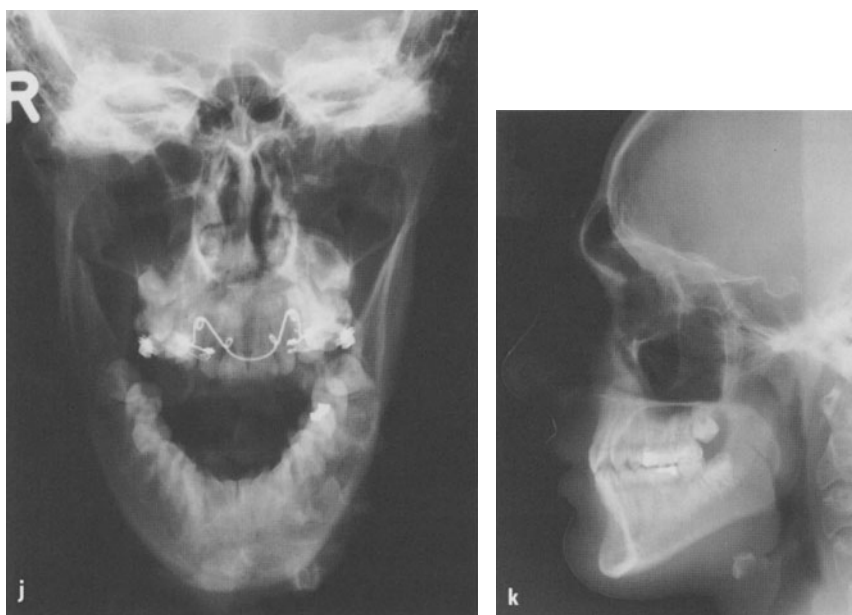


Fig. 53j-k. Unilateral H:E., slender-non-slender type, still active. **j** The mandible p.a. projection at maximum mouth opening looks very similar to that of a H:E., slender type case, except that in the non-slender type the whole mandible is often much stronger. **k** The lateral cephalogram is unrevealing of any possible anomaly and therefore of little value in diagnosing the H:E. anomaly

served down to a depth of about 2.9 mm, the presence of cartilage islands throughout the spongiosa of this specimen does not need to be abnormal. Similar to the situation in another case of H.E. (Fig. 48u), marrow spaces of the spongiosa were conspicuously large, while the thin trabeculae disclosed a strictly oriented, nearly parallel arrangement (see Fig. 79e).

Diagnosis. Actively growing condyle exhibiting a conspicuous spongiosa characterized by few slender, almost parallel trabeculae.

Comment: Similar to that of the other case of H.E. (Fig. 48u), this specimen did not show any signs of enhanced condylar growth, whereas the appearance of the subchondral spongiosa was typical of H.E.

Discussion. In evaluating the amount of lateral shift of the mandible in cases of unilateral H.E., one should not rely only on the position of the lower teeth midline compared with the upper midline. One must always compare the upper as well as the lower teeth midline with the facial midline. This latter almost always coin-

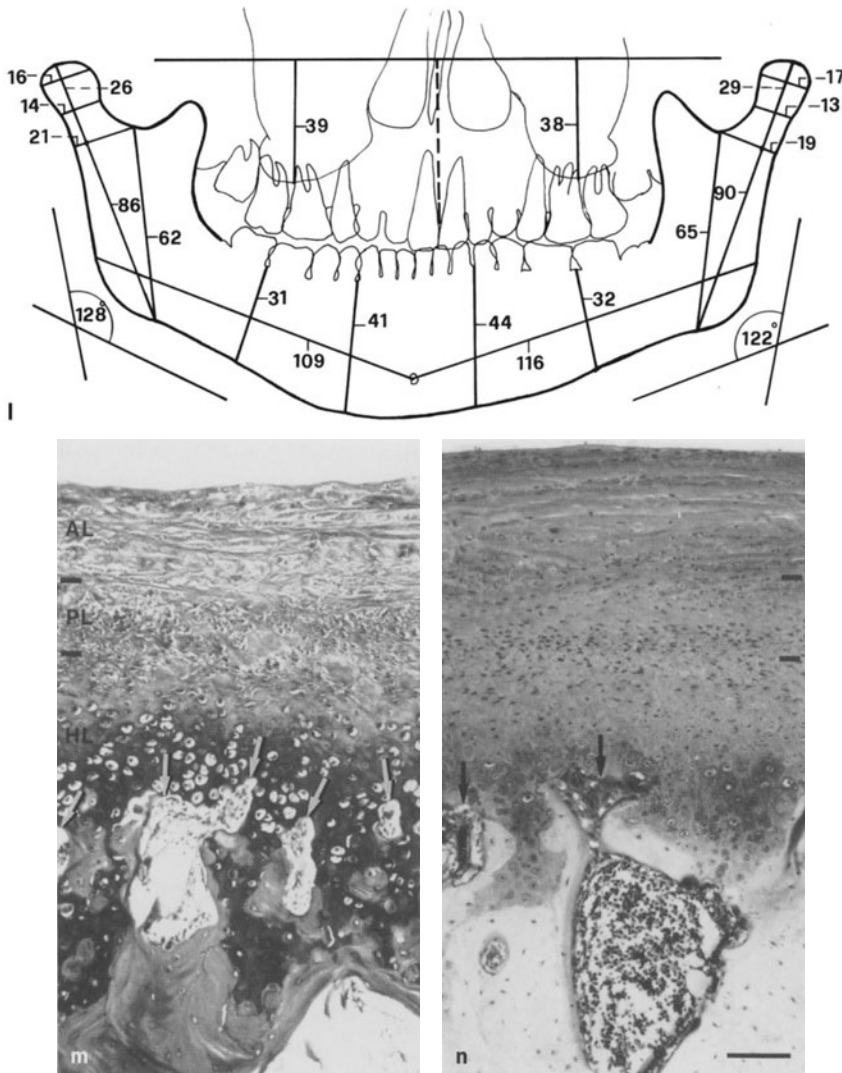


Fig. 531-n. Unilateral H.E., slender-non-slender type, still active. **I** The tracing of the panoramic view and its measurements clearly prove the H.E. anomaly and also its difference from the slender type. **m, n** Microscopic appearance of the articular tissue of the excised condyle **m** in comparison with that seen in a normal specimen from a male of about 16 years of age **n**. Whereas the cartilage in the hyperthrophic layer **HL** of the affected condyle **m** is clearly hypertrophic, cartilage in the control specimen **n** exhibits relatively small chondrocytes and wider matrix septa of less metachromatic staining. Also, lacunae in the zone of cartilage erosion (*arrows*) are more numerous and larger in the affected as compared with the normal condyle. (*AL* articular layer, *PL* proliferative layer; **m** paraffin section, **n** plastic section, toluidine blue, original magnification $\times 130$, bar = 100 μm)

cides with the position of the nasal septum, except in those cases in which the maxilla has been deformed or displaced due to trauma, surgery or any possible congenital or acquired anomaly such as secondary cleft lip and palate deformities, etc. The crista galli can always be recognized in the p.a. projection of the mandible.

Making the diagnosis from the panoramic view only, this case of unilateral H.E. seemed clearly to be of the non-slender type. But the TMJ–ramus tomograms disclosed that this case was not a pure non-slender nor a pure slender type of H.E. It is a deformity somewhere in between.

This case also proved that to enable a proper diagnosis, several standard radiographs should be requested routinely.

For treatment, such a diagnosis was not very important, as long as there was no real stretching of the angle. In this case it did not matter where and in which direction the lateral cortical cut of the sagittal splitting procedure was placed. If the angle was really stretched, then it should be mandatory to create a better angle, either by leading the lateral cortical cut directly to the angle area or by the use of any other surgical procedure which is able to improve the angle area. Although the boy was less than 16 years old and the end of his skeletal growth was rather close, in spite of this, a high condylectomy seemed to be indicated in a case with such a severe mandibular asymmetry, in order to prevent further growth on the affected side.

Unilateral H.E., Non-Slender Type and Ipsilateral Hemifacial Hypoplasia (Fig. 54)

Sex and Age. Female, 18 years and 7 months.

Aetiology and History. The mandibular asymmetry had developed gradually during pubertal growth. The patient did not remember any possible cause nor any similar anomaly in the family. The patient wished correction for aesthetic and occlusal reasons. She had been under orthodontic observation in her very distant home country since she was 11 years and 5 months of age.

Clinical Findings. From in front and in the profile view (Fig. 54a, b), the young lady presents a rather long face with mandibular asymmetry, with the chin slightly over to the left as well as the left corner of the lip. The lower lip is slightly protruding and there is a longer flat cheek, zygoma and mandibular region on the right side, while the left is clearly more voluminous, in particular the horizontal ramus and angle area. Mouth opening was normal.

In occlusion (Fig. 54c–e) the right side has a class III relationship and the left is in crossbite with a gap in the canine region.

The histological examination of the resected part of the condyle did prove the existence of a H.E., but could not define any ongoing growth hyperactivity, in spite of the positive scintigraphic report, similar to the case of Fig. 48.

Conclusion. For proper diagnosis it is often necessary to have several standard radiographs at hand, including TMJ–ramus tomograms. The panoramic view is not the only mandatory one. In severe cases it is also mandatory to have scintigraphic information about the cellular activity in the two condyles. If in such cases condylar hyperactivity is clearly occurring, immediate high condylectomy is indicated, while in less severe elongation cases one may wait until growth is complete and then perform the necessary corrective surgery without any operation on the joint.

In evaluating the amount of sideways shift of the chin and the teeth midline it is necessary to compare their positions in relation to the facial skeletal midline and not to that of the upper teeth only. Generally, but not invariably, the position of the nasal septum coincides with the facial midline, but the position of the crista galli is the final determinant.

Histology seems to be able to diagnose a H.E. when growth is still active, but not when the hyperactivity has ceased.

Radiographs. The panoramic view (Fig. 54f) shows an almost normal mandible but with a clear elongation of the right side. The condyles are of normal shape and size and so is the left angle, while the right angle is clearly stretched (8° difference). The mandibular body in the molar area on the right side is less high than on the left. The lower teeth midline seems to be shifted to the left by almost the width of a lower incisor. The chin is rather high (deep) and its prominence is also shifted to the left by the same amount compared with the midline of the upper teeth. The distance from the angle to the chin midline seems remarkably greater on the right side than on the left. The measurement on the tracing proves that the difference was 3 mm only. The upper teeth midline remain in the facial midline.

The TMJ–ramus tomograms (Fig. 54g, h) show a normal condyle with a rather short neck and normal angle on the left side. The right condyle is also normal in size but with a longer neck and the angle of that side of the mandible is stretched. There are no signs of skeletal hy-

poplasia which could have explained the rather flat right side of the face.

The mandible in p.a. projection after 3 years of orthodontic treatment (Fig. 54i), shows the asymmetry in length very clearly and also the midline of the maxilla to be exactly in the midline of the face.

The lateral cephalometric projection showed a clear elongation of the facial lower third, attributable to both jaws (bimaxillary long face).

The hand radiograph showed that all epiphysal cartilages of the fingers were completely closed, whereas at the radius it was still just recognizable.

The Water's view taken before surgery, revealed a clear difference between the right and left zygomas. The hypoplasia of the right middle third was clearly obvious by the fact that the left infraorbital rim was lower down than the right and the left maxillary sinus was much smaller than the right.

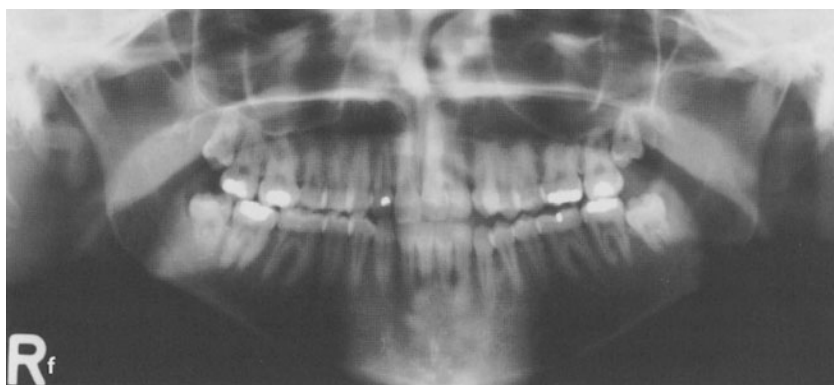
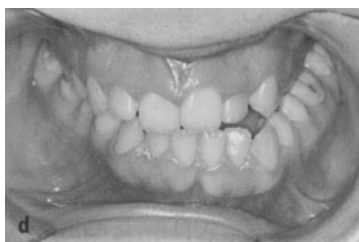


Fig. 54a–f. Unilateral H.E., slender/non-slender type and ipsilateral hemifacial hypoplasia. **a, b** The patient's appearance at age 18 presenting facial asymmetry not only due to unilateral H.E. but additionally also due to a bimaxillary long face anomaly. **c–e** Typical occlusion abnormality of strongly growing unilateral H.E. **f** The presurgical panoramic view showing a H.E. of slender/non-slender type, on the right side

The measurements on the tracing of the panoramic radiograph (Fig. 54j) produce rather disappointing results. The right horizontal ramus is only 4 mm longer than the left and the right ascending ramus measures 3 mm more in length than the left. But there is quite a difference between left and right angles, the latter being 8° larger than the left. Of some importance for a H.E. is, additionally, the reduced height of the horizontal ramus in the molar region, 4 mm compared with that of the left side. All these differences together make for a diagnosis of H.E. of the right side, although the overall picture does not look very impressive.

Provisional Diagnosis. Unilateral H.E., slender/non-slender form on the right side accompanied by a clear

hypoplasia of the same half of the face and a bimaxillary long face.

Treatment Plan. Combined orthodontic and surgical treatment. The model operation suggested that in spite of presurgical orthodontics for 3 years, the maxilla must be surgically widened by 6 mm in the posterior palate area, raised by 5 mm and the mandible brought into occlusion using bilateral sagittal splitting of the rami (Fig. 54k). Any residual facial asymmetry should then be corrected as required, in a second intervention.

Actual Treatment. The presurgical orthodontic treatment was carried out in the young lady's home town since she lived in another country. The orthodontist had

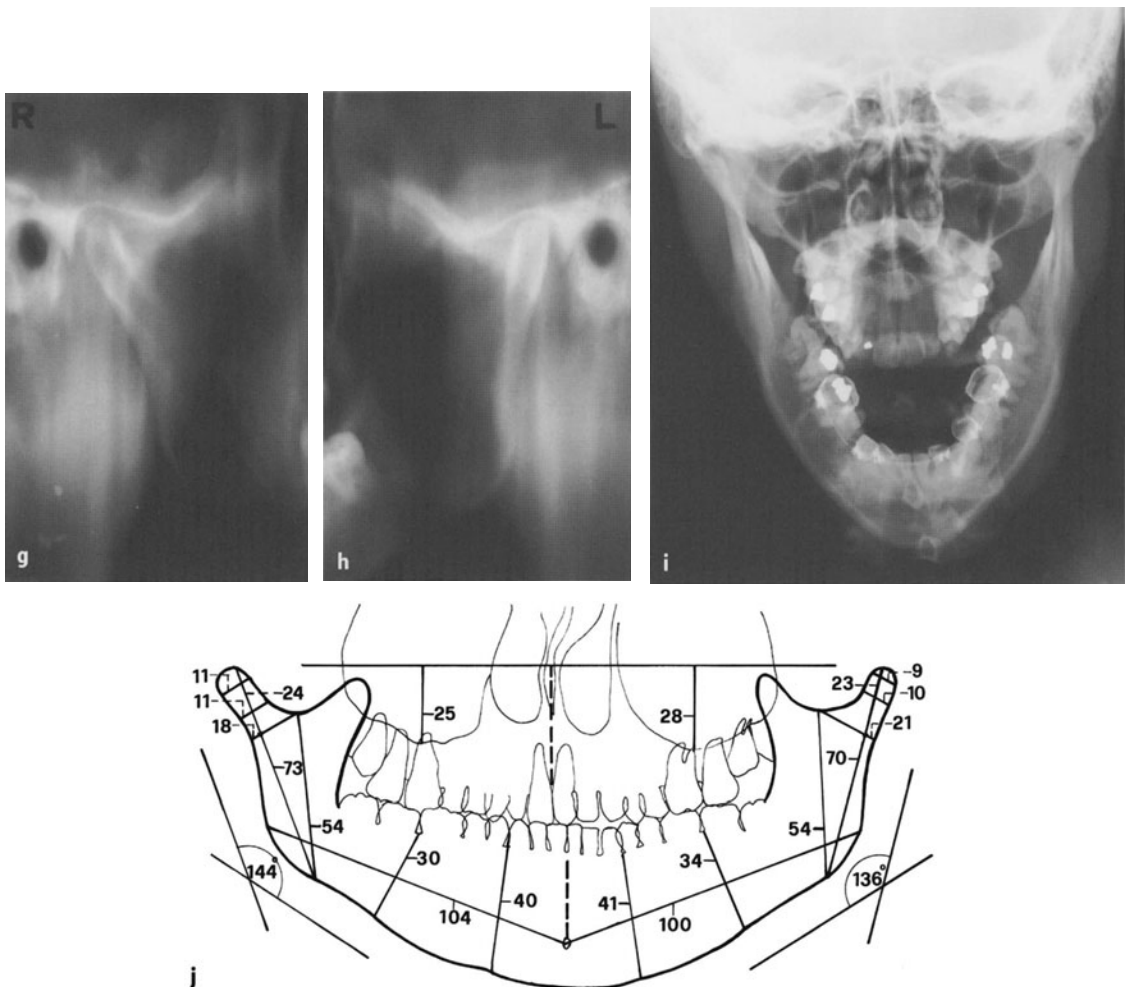


Fig. 54g-j. Unilateral H.E., slender/non-slender type and ipsilateral hemifacial hypoplasia. **g, h** The tomograms of the TMJ-ramus regions show a clear elongation of the right neck compared with the left and a clear stretching of the angle on the right side. **i** The mandible p.a. projection permits the facial midline to be recognized from the crista galli down, via the nasal septum, to the maxilla and the mandible. It shows a clear elongation of the right hemimandible. **j** The tracing of the panoramic view discloses that the differences between the left and right hemimandibles are rather small

extracted the first bicuspsids on each side of the mandible 10 months before surgery. When she came for surgery, the two arches of teeth could not be brought into proper interdigitation. In spite of that, it was agreed with the patient and the orthodontist that we would correct the situation surgically, and he should complete the orthodontic treatment postoperatively.

At a first operation, the maxilla was widened by 6 mm and repositioned 5 mm higher and fixed there; and after bilateral splitting of both rami the mandible was fixed in the best occlusion possible. The chin prominence was trimmed on the left side using carbonite burs but 8 months later there was still unacceptable facial asymmetry. This was not due to the deviation of the mandible to the left as before surgery, but due to a bony surplus at the angle on the left side and a lack of bony and soft tissue contour on the primarily elongated right side (Fig. 54m).

The postsurgical occlusion was the best achievable result on the basis of the prerequisites after 3 years of presurgical orthodontics (Fig. 54n-p).

Because of the still existing facial asymmetry, a second operation was planned (Fig. 54l) and carried out. The height of the chin was reduced by a slice ostectomy, the flatness of the elongation side was corrected by subperiosteal implantation of slices of rehydrated lyophilized cartilage along the whole of the right side of the mandible and in the right zygomatic area. In addition, the protruding contralateral side was reduced by a rather radical decortication of the left angle and body region. A satisfactory symmetrical result was achieved (Fig. 54q,r). The occlusion still needed some further orthodontic attention.

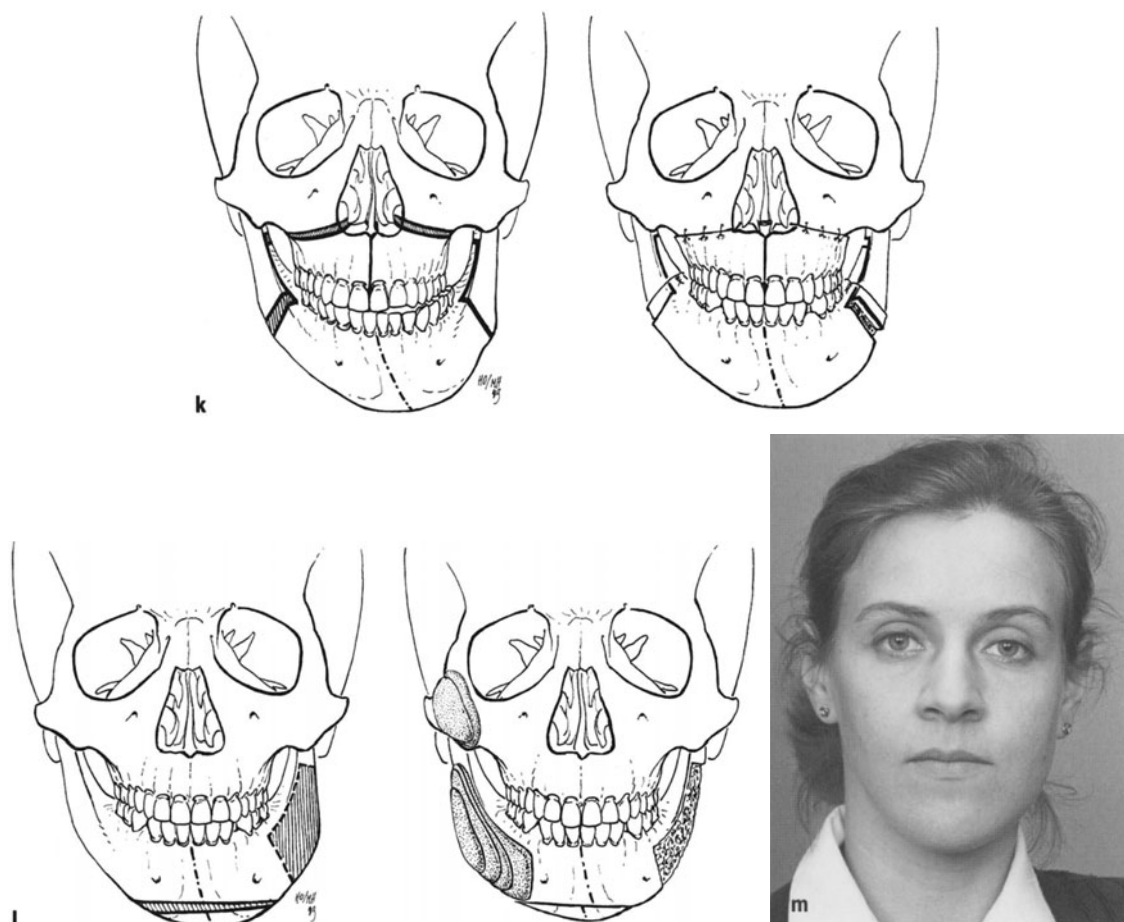


Fig. 54k-m. Unilateral H.E., slender/non-slender type and ipsilateral hemifacial hypoplasia. **k, l** Schematic drawings of the planned corrective surgery: in a first operation **k** rotation of the lower half of the facial skeleton for correction of the occlusion and reduction in facial height. In a second operation **l** additional correction of the remaining skeletal asymmetry. **m** After correction of the H.E., the hypoplasia of the same side and the surplus of the angle area of the other side becomes more visible



Fig. 54n–r. Unilateral H.E., slender/non-slender type and ipsilateral hemifacial hypoplasia. **n–p** These occlusal pictures show the result of the correction of the H.E. anomaly and also the need for more orthodontic treatment. **q, r** The patient's front and profile appearance 9 months after the second operation

Discussion and Conclusion. In a rather remarkable percentage of unilateral H.E. cases, there is a flatness of the face on the same side, in addition to the elongation which is due to the shape of the ramus and body and also the soft tissues. Occasionally, the zygoma and zygomatic arch area is also hypoplastic. As in this case the

chin prominence may be further over to the opposite side than the teeth and the contralateral mandible may be more voluminous than normal. This requires an additional correction, not just repositioning of the mandible into proper occlusion.

13.2.4

Differential Diagnosis of Unilateral H.E.

There is not much possibility of diagnosing a case as a unilateral H.E. when it is something else. The most common mistake is to diagnose a unilateral hemimandibular hypoplasia as a contralateral H.E. As we generally do not know what the pattern of the patient's mandible would be if there were no asymmetry, it is often not that easy to say which side is the normal one. In anlage-induced antemandibulism, a unilateral hypoplasia may easily be diagnosed as a contralateral H.E. The same is true for misdiagnosing an asymmetry when the case is a normomandibulism but suffers from an unilateral hemimandibular hypoplasia. On the other hand, a case of retromandibulism with a unilateral H.E. may be

wrongly diagnosed as a contralateral hemimandibular hypoplasia.

H.E. does not displace the corner of the mouth on the affected side upwards or downwards, but forwards only. Hemimandibular hypoplasia always develops before growth has ceased. For that reason, the maxilla cannot grow downwards equal to the normal side. As a consequence, the corner of the mouth is always higher and further back on the hemimandibular hypoplasia side than the contralateral side.

There are cases in which even scintigraphy does not elucidate the problem. Finally only histology of the condyle of the longer side will help to make the diagnosis clear (see case Fig. 51).

13.3

Bilateral Hemimandibular Elongation

As there is no doubt that a unilateral H.E. is caused by hyperactivity of the condylar L-regulator on one side, there is no reason why the L-regulator should not become hyperactive on both sides. We assume that unilateral hyperactivity is not due to a central steering mechanism. Otherwise both sides would react equally as in acromegaly. Therefore, we have to accept that both sides can simultaneously, or independently start as hyperactivity of the L-regulator. Since that start has no control mechanism, neither in time nor in degree of hyperactivity, the two sides will react differently, the one side producing more length than the other. This means that in every case of asymmetrical antemandibulism (class III mandibular position), with or without an open bite, we have to consider it a result of a bilateral H.E. process. Of course, there may be the very rare case in which both sides behave the same thus producing a symmetrical bilateral elongation. In anlage-induced antemandibulism, in my opinion, there is no reason why an asymmetric

situation should be produced. Therefore, any asymmetrical bilateral elongation of the mandible is a result of bilateral hyperactivity of the condylar L-regulator. There is another point which makes me believe this. This is the fact that we can find on one side a slender type and on the other side a non-slender type of a H.E. in such an asymmetric, bilateral elongation case.

There is another fact which can make things even more difficult for diagnosis. While in the real antemandibulism case both sides elongate equally, because of the genetic steering influence, there is still the possibility that during growth or even when growth has already ceased, a unilateral condylar growth hyperactivity may be superimposed on such an antemandibulism case by one of the three hyperactivity types (H.E., H.H. or hybrid form). In such a case the less experienced might easily make the mistake of diagnosing it to be the result of bilaterally unequal condylar growth hyperactivity.

The following cases are presented to attempt to demonstrate what a variety of intermaxillary relationships and external appearance bilateral H.E. can produce, according to my theory.

Bilateral H.E., Slender Type, Active During Puberty (Fig. 55)

Sex and Age. Female, 15 years and 7 months.

Aetiology and History. The girl's parents did not know any possible cause for the forward growing mandible, neither was such an occurrence known in the family history. The chin started to move noticeably forwards at the age of about 11 years. Her dentist extracted the first upper bicuspid on each side. He suggested that she waited to see what would happen. When the girl was 15 years and 7 months old she was referred to me for evaluation and treatment planning. Because of the girl's age it was suggested that she waited until growth had ceased.

When she was 18 years and 8 months there had been no further forward growth of the mandible for almost 1 year. But from 15 years and 7 months until 17½ years there was markedly further forward growth of the mandible with increase in the lateral shift to the right side.

Clinical Findings. At the age of 15 years and 7 months the facial lower third is clearly asymmetrical. It appears to be composed of two unequal sides (Fig. 55a, b). The mandible is protruding forward beyond the maxilla. The chin is slightly out of the midline over to the right side. The right angle region is more voluminous than the rather slim and flat left side and also more voluminous than normal. Both angles are stretched, the left more so than the right. The volume surplus in the right angle region originates essentially in the musculature. Mouth opening was unaffected. The occlusion shows

the picture of a bilateral, asymmetrical antemandibulism. In the maxilla the first bicuspid is missing on each side.

Three years later, at age 18½, the face still looks to be composed of two unequal halves (Fig. 55c, d). The mandible now seems longer and is more prominent. The left side is flat and straight. But the right side now looks less voluminous. The lip commissure is horizontal with its left corner further back than the right. The mid-facial areas are rather flat. The patient presents the typical appearance of an asymmetric antemandibulism.

With the teeth in the closed position, there is a full class III occlusion in the canine relationship on both sides (Fig. 55e–g). The midline of the lower teeth is over to the right side by 3 mm – a bit more than 3 years earlier. There is a negative overjet of 3 mm. There is no tilting of the teeth to either side.

Radiographs. The panoramic view of the mandible in the closed position at the age of 15 years and 7 months only proved what had been found by clinical investigation. Both sides of the mandible had a clearly elongated condylar neck and a stretched angle, the left side more than the right. The lower midline was over to the right side by the width of half a lower incisor. The whole mandible seemed very gracile. Three years later (Fig. 55h) the mandible appears stronger, but there was no change

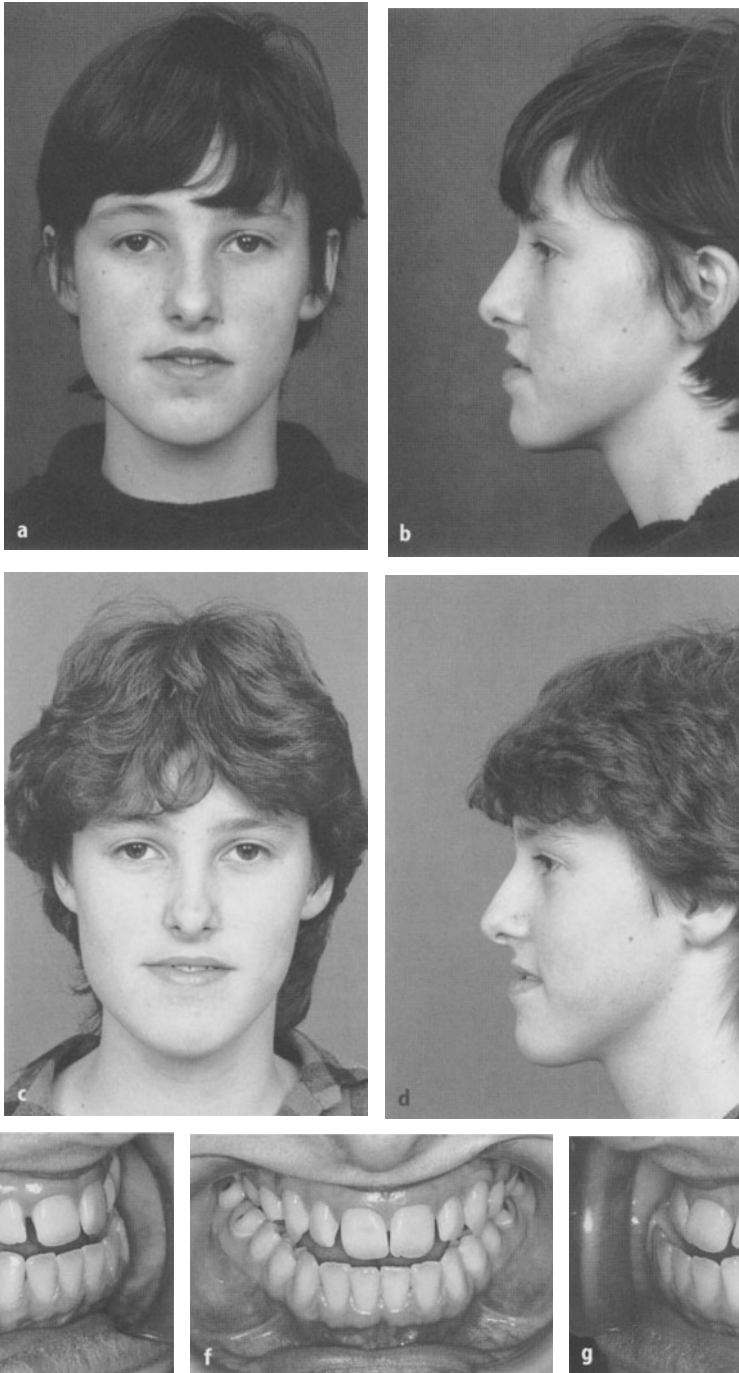


Fig. 55a–g. Bilateral H.E., slender type, active during puberty. **a, b** The patient's front and lateral views at age 15 1/2 years demonstrating a clinical picture of asymmetrical antemandibulism. **c, d** The patient's appearance 3 years later. **e–g** The patient's occlusion is typical of such an anomaly

in its shape except the left condylar neck appears to be clearly slimmer than the right.

The measurements of the tracing of the panoramic radiograph (Fig. 55i) reveal interesting facts: The right as-

ending ramus is 4 mm longer than the left and the horizontal ramus is 7 mm shorter than the left. The right articular process measures 10 mm more in length than does the left. The left angle is 3° greater than the right,

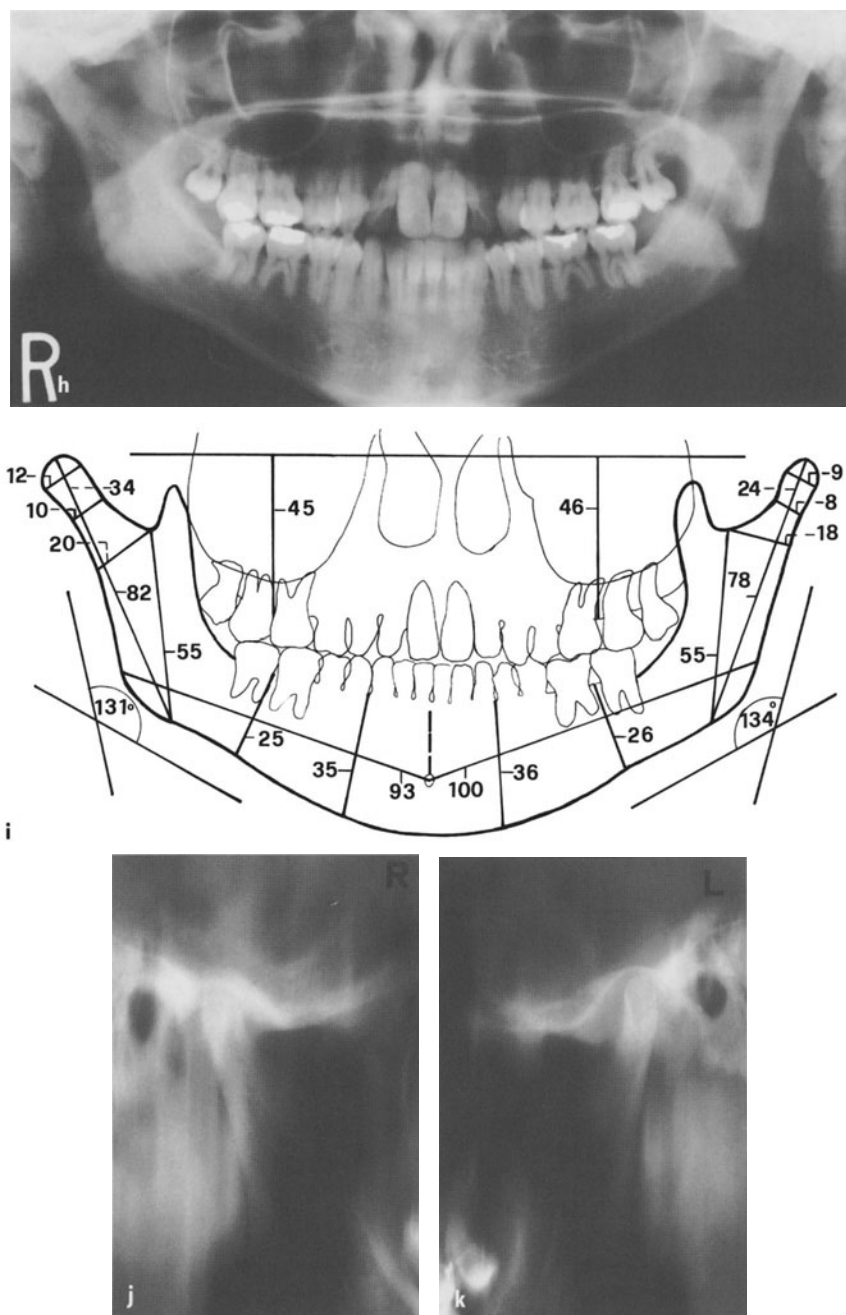


Fig. 55h–k. Bilateral H.E., slender type, active during puberty. **h** The panoramic radiograph discloses the two long articular processes with small condyles and stretched angles and the resulting asymmetry. **i** The measurements of the tracing of the panoramic radiograph show that the differences between the two sides are larger than expected. **j, k** The TMJ–ramus tomograms show very clearly that both condyles are rather small compared with normal. They also show the very long slim necks on both sides

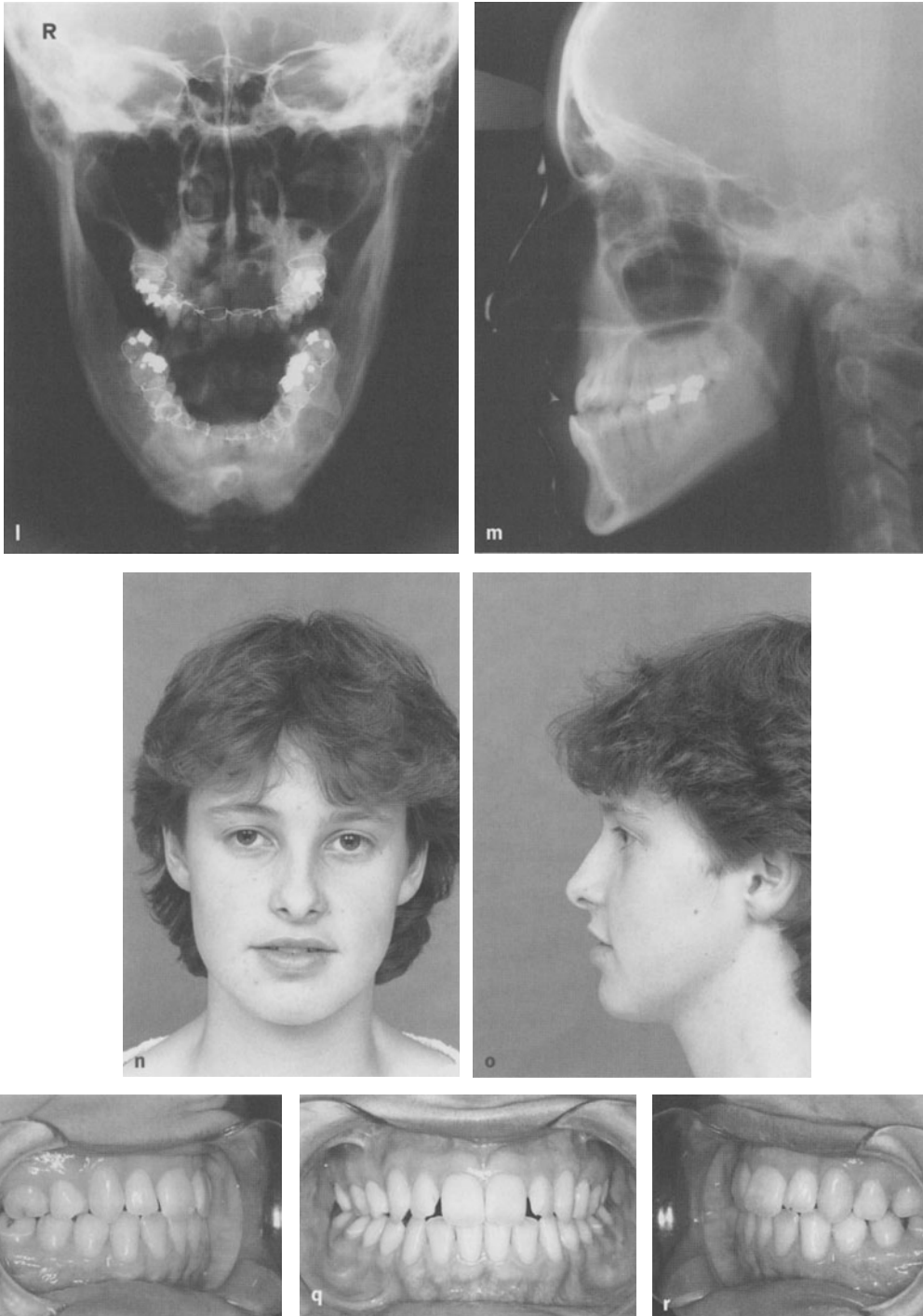


Fig. 551-r. Bilateral H.E., slender type, active during puberty. **l** The mandible in p.a. projection also discloses the very long and slender hemimandibles on each side. But in addition it discloses that both sides are not equal in length. **m** The lateral cephalogram shows the rather long middle third of the facial skeleton as the reason for the patient's flatness in the mid-facial area. **n-r** The patient's appearance and occlusal situation 1 year after surgical repositioning of both jaws in accordance with the planning

being 134° – definitely a stretched angle. These figures indicate that the hemimandible with a longer horizontal ramus will not automatically have a longer articular process and ascending ramus.

The TMJ–ramus tomogram (Fig. 55j,k), taken at age 18 and a half years, shows a rather small right condyle, with a rather long neck and a broad base. The left condyle is clearly smaller than the right and has almost no greater p.a. diameter than the very thin and elongated neck.

The mandible in p.a. projection in maximum opening (Fig. 55l), taken at the same time, shows a slight asymmetry of the gracile mandible with the left ramus-body region slightly longer than on the right and more gracile. The right mandible looks stronger than the left, particularly in the angle and horizontal ramus region. The symphysis is shifted slightly over to the right side.

The lateral cephalogram (Fig. 55m) discloses clearly the stretched gracile mandible in a negative overjet position and with a maxilla of normal sagittal length, in a normal position for a vertical face profile.

Treatment Plan. According to the model operation a very good occlusion should be achievable without any presurgical orthodontics. Therefore, the plan was a bilateral sagittal splitting procedure on the ascending rami and mandibular repositioning combined with upwards repositioning of the maxilla by 4 mm and advancing it by 5 mm.

Actual Treatment and Result. The surgery was carried out as planned, by a trainee and the result achieved (Fig. 55n, o) is in accordance with the planning, in the patient's ap-

pearance as well as in the occlusion (Fig. 55p–r). No pre- or postsurgical orthodontics was carried out.

Discussion. This straight, bilateral, slender type H.E. case was handled in accordance with our experience. Wait until growth has ceased and then plan the treatment by surgery alone and if the model operation permits this, a good occlusion is to be expected. Normally, a bilateral H.E. comes to a standstill in its hyperactivity by the age of 17–18 years, sometimes a year earlier and sometimes a year later. Of special interest is the fact that the more elongated side of a bilateral H.E., slender type, produces a slimmer and longer condylar neck than that on the opposite side and it also reduces the p.a. width of the condyle to almost that of the very gracile condylar neck.

Conclusion. The bilateral H.E., slender type, of moderate degree, constitutes no diagnostic difficulties. It can normally be managed in the standard way. If one waits until the abnormal mandibular growth is complete, bilateral repositioning of the mandible can produce a very pleasing occlusal and aesthetic result, if necessary with an additional advancement of the maxilla, when there has not been a negative influence on the teeth position by some rather unsuccessful orthodontic manoeuvres. The teeth are generally in such a position that the surgeon only needs to set the mandible into the planned occlusion. In general, there is practically no tilting of the teeth in a bilateral H.E. case, contrary to the unilateral H.E. case.

Bilateral H.E., Slender Type, Mild Degree (Fig. 56)

Sex and Age. Female, 17 years of age.

Aetiology and History. The mother informed us that the girl's grandfather had the same asymmetrical "prognathism" and also her cousin had a similar appearance but less pronounced than that of the girl. At the age of 6 years the mother realized that the lower teeth midline was slightly to the left. Very slowly the situation became worse with the chin shifting over to the left side. At the age of $15\frac{1}{2}$ the girl wanted to have the problem corrected. She consulted an orthodontist who suggested waiting until growth had ceased. When she was 17 and 4 months he had commenced presurgical orthodontics. The girl was operated upon at the age of $18\frac{1}{2}$ years.

Clinical Findings. The 17-year-old girl has a long slim face with the chin midline deviated to the left by a few millimetres only (Fig. 56a, b). The chin prominence is rather pointed. There are almost no angles of the jaw.

The mid-facial region is flat, almost concave. Mouth opening is normal with the chin returning into the facial midline.

Almost all of the lower teeth are tilted lingually, in particular the front teeth. Their midline is shifted over to the left by the width of half a lower incisor when compared with the midline of the upper teeth. There is a bilateral class III occlusion in the molar region but there is still a very slight overbite anteriorly (Fig. 56c).

Radiographs. *The panoramic view* (Fig. 56d) reveals the very typical shape of a bilateral H.E., slender type. On both sides, the condyles are small, their necks slim and long, on the left less so than on the right side. Both angles are stretched. The right ascending ramus is longer than the left and it has an almost straight posterior border. The left is closer to normal, with a flat posterior

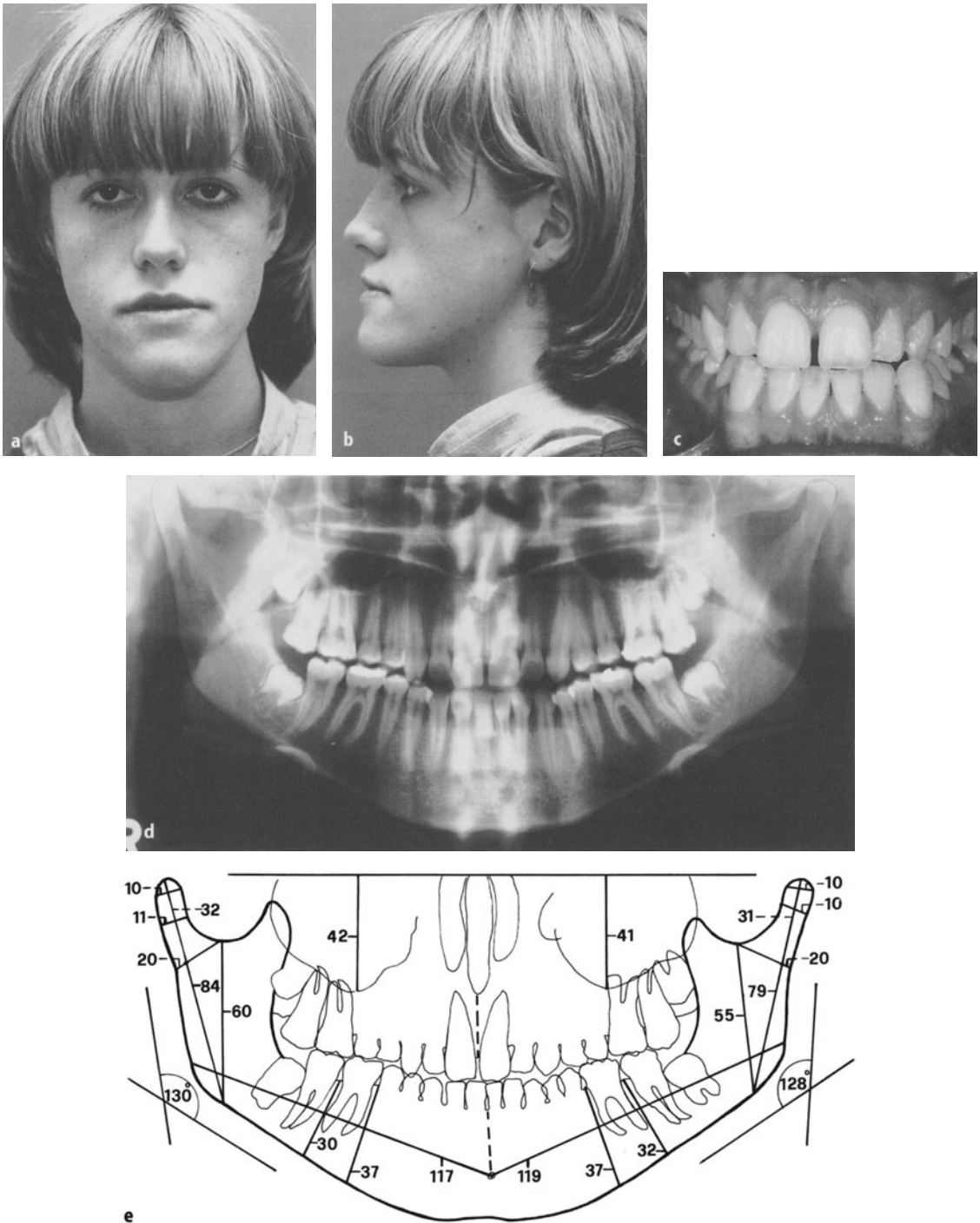


Fig. 56a–e. Bilateral H.E., slender type, mild degree. **a–c** The patient's front and profile views and occlusion at 18 years. **d, e** Typical panoramic view of bilateral H.E., slender type, with more elongation and angle stretching on the right side, also confirmed by the measurement of its tracing

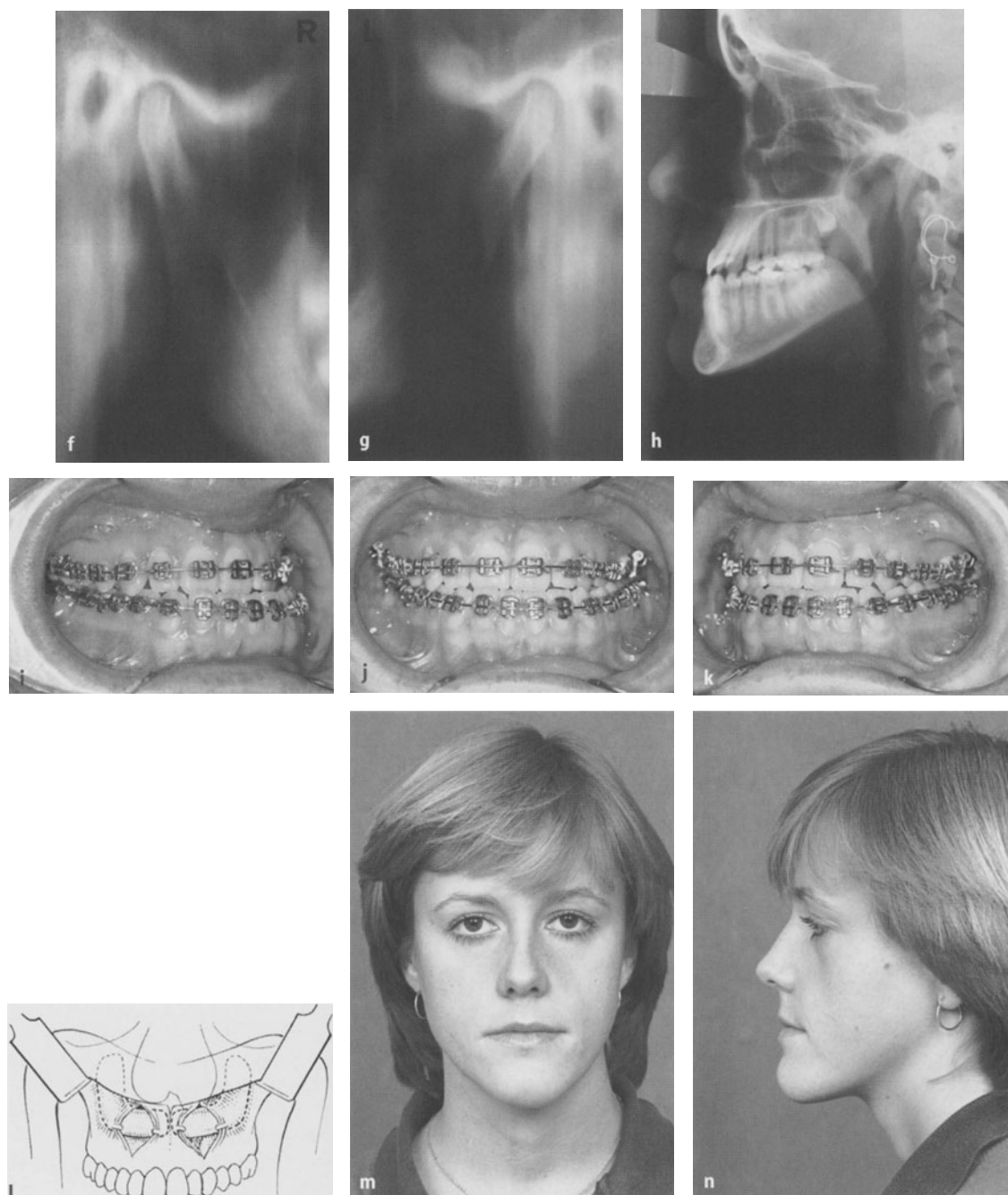


Fig. 56f-n. Bilateral H.E., slender type, mild degree. **f, g** The TMJ-ramus tomograms confirm in detail the ramus and condyle situations on the two sides. **h** The lateral cephalogram discloses not only the long horizontal rami but also the flatness in the paranasal/mid-facial regions. **i-k** The patient's occlusion after presurgical orthodontics. **l** Diagrammatic illustration of paranasal onlays for correction of flat paranasal areas. **m, n** The patient's front and profile views 2 years after surgery. The flat angle regions have been clearly improved by the sagittal splitting technique with the lateral cortical cut towards the angle and so was the paranasal flatness by paranasal lyo-cartilage onlays



Fig. 56o–q. Bilateral H.E., slender type, mild degree. **o–q** The patient's final occlusion, also 2 years after repositioning of the mandible

concavity and a somewhat rounded angle, although, also very open. The right horizontal ramus is slightly longer than the left. Both sides show very little distance between the mandibular lower border and the apices of the second and third molars.

Measurement of the tracing (Fig. 56e) of this radiograph does not reveal large differences between the right and left sides of the mandible. The horizontal rami are almost equal in length and height. The two articular processes are almost equal too. The total length of the ascending rami differs by 5 mm only, due to the difference in length of the two trunks, in favour of the right one. The angles are also almost the same. Both are within the range of normal, the right measuring 130° and the left 128°.

The tomograms of the TMJ and rami (Fig. 56f,g) confirm what was seen on the panoramic view. The right articular process is longer and slimmer than the left. This is also true for the condyle only.

The p.a. projection of the mandible in the maximum opening position showed the mandible to be back into the facial midline with a clearly longer right ascending ramus than the left one which also seemed to be elongated. The lateral cephalometric view (Fig. 56h) also confirms what had been found to be abnormal in the panoramic view: a long mandible with long condylar necks, lingually tilted front teeth, but not a real negative overjet and a maxilla normal in size and position, but rather hypoplastic zygomatic infraorbital regions.

Provisional Diagnosis. Bilateral mild degree H.E., slender type, and hypoplasia of the zygoma-infraorbital-paranasal regions.

Treatment Plan. The case was discussed with the girl's orthodontist (Dr. P. Stöckli). Presurgical orthodontics would be necessary in order to bring the axes of the teeth into proper angulation to the jaw base and to create dental arches which would fit together. After the presurgical orthodontic treatment (Fig. 56i–k), bilateral sagittal splitting of the rami and mandibular repositioning should provide a normal occlusion and paranasal onlays of lyophilized human cartilage would additionally correct the mid-facial flatness (Fig. 56l).

Actual Treatment. The presurgical orthodontic preparation was done within 13 months. Then the surgery was carried out according to the plan, using perimandibular wires for the split rami and 6 weeks intermaxillary fixation. In addition an L-shaped piece of rehydrated lyophilized cartilage was placed subperiosteally in the paranasal region of both sides. The result of the correction, checked 1 year and 9 months after surgery, is very satisfactory, in both the patient's appearance (Fig. 56m, n) as well as in the occlusion (Fig. 56o–q).

Discussion and Conclusion. H.E. may develop unilaterally or bilaterally. The unilateral as well as the bilateral cases may be seen in very mild to excessive degrees. This was a non-problematic case, easy to diagnose and also to treat. When it is of a bilateral mild degree, the mandible will come forwards on both sides, but not very much. Through lip pressure, even a slight overbite of the front teeth can be produced spontaneously. The worst thing to do in such a case is to extract a tooth on each side and try to move the teeth backwards. It will never correct the external appearance but it makes things very difficult for the surgeon. Sometimes I had to ask the orthodontist to move the orthodontically retruded teeth back into proper angulation to the jaw base, reopening the spaces of the extracted teeth. Only then could the surgeon correct the asymmetrical slight antemandibulism.

In some cases, the diagnosis of a bilateral H.E. can be made by just seeing the patient's face and occlusion. In other cases one will need radiographs for exact diagnosis. These radiographs are always interesting to look at as the pure uni- and bilateral H.E. slender types have such a typical shape of the whole mandible, including its condyles and necks.

Whether or not these mild cases start with the forward growth from a genetic class II position or a class I or III is very difficult to say. It is obvious that the same amount of forward growth due to bilateral L-regulator hyperactivity does produce a mild or a severe overjet, depending on the primary genetic pattern of the mandible.

Bilateral H.E., Slender Type, Producing Severe Asymmetric Open Bite (Fig. 57)

Sex and Age. Male, 20 years of age.

Aetiology and History. The asymmetry of the chin became obvious at 12 years of age and the final asymmetric open bite situation was reached by the age of 19 years. The patient had no explanation for why it had started. Nothing similar was known in his family history. He came for improvement in his chewing capability and of his outward appearance. Twenty three years later the patient has four almost grown-up children, none of them has mandibular asymmetry or symmetrical antemandibulism.

Clinical Findings. The patient has a mandibular long face with an asymmetry to the left, and a rather high (deep) and slightly retruded chin (Fig. 57a). The lip commissure ascends slightly to the left side, with the left commissure further posterior and higher than the right. Mouth opening is within the normal range, with much less asymmetry of the chin than when in the closed position.

There is a circular open bite with occlusion of the last molars only (Fig. 57b). The midline of the lower teeth is shifted over to the left by the width of two lower incisors.

Radiographs. The panoramic view did not exist in the days when the patient came for treatment.

The lateral oblique views of the two halves of the mandible (Fig. 57c, d) disclose that both sides have a very stretched angle and thin and long condylar necks.

The p.a. projection of the mandible in the closed position (Fig. 57e) shows a remarkable length difference between the two sides, the right being much longer than the left.

TMJ radiographs were not then routinely requested.

The lateral cephalometric view (Fig. 57f) discloses the straight shape of the mandible with almost no angle and long thin condylar necks. There is slight retromaxillism. The open bite increases the height of the lower third much more than does the actual skeletal situation. There is no real hypergenia, but also no real well-formed prominence of the chin.

Provisional Diagnosis. Bilateral asymmetric H.E. slender type, producing a severe asymmetric open bite.

Treatment Plan. According to the model operation (Fig. 57g), a very satisfactory occlusal result could be achieved by bilateral sagittal splitting of the rami and mandibular repositioning. As the midline of the lower incisors coincides with that of the symphysis, the mandibular asymmetry will be corrected simultaneously (Fig. 57h).

Actual Treatment and Result. The typical sagittal splitting of the rami procedure was applied on both sides, with the lateral cortical cut from behind the first molars down to rather in front of the angle (Fig. 57i) (in 1962). Two circumferential wires and intermaxillary fixation for 6 weeks secured the position of the segments. There were no complications. The occlusion and the appearance resulted as planned (Fig. 57j, k).

Twenty two years after surgery the panoramic view (Fig. 57l) still reveals the former shape of the two condylar necks and condyles. The right one is quite a bit longer than the left. Both condyles are much smaller than normal, the left even more so than the right. It shows some evidence of destruction, with a subcondylar anterior exophyte. There is also no change in the patient's appearance nor in the occlusion (Fig. 57m, n).

Discussion. A H.E. can either shift the mandible forwards and over to the other side producing a crossbite only, or it can also push the mandible downwards producing an asymmetric open bite. When both sides are involved in the same way then they are generally not equally strong, resulting in an asymmetric open bite. We know by experience that the correction of such a severe open bite as in this case will relapse unless the maxilla is also mobilized and raised in its posterior part. This is definitely true when the open bite has developed due to muscular activity which we do not eliminate with a bilateral sagittal splitting correction. But if the open bite has developed due to misregulation of condylar growth as in this case, which has either been eliminated by a high condylectomy or has come to a standstill by itself, then there should be no danger of relapse. The inactive intramandibular cause of the open bite cannot produce an open bite or an asymmetry again unless it is reactivated. This case seems to prove this theory.

Conclusion. Bilateral H.E. will generally produce an asymmetry of the mandible either in an antemandibulism position only, or with an additional open bite. Since the cause of the anomaly is hyperactivity of one or both condylar growth regulators the correction should not be performed unless the hyperactivity is no longer present. When the cause of the anomaly no longer exists then the correction of its skeletal abnormality will be safe. This fact is in congruence with the following author's principle: "*The cause of a given anomaly should either be inactive or eliminated prior to definitive surgery*" (Principle No. 29).

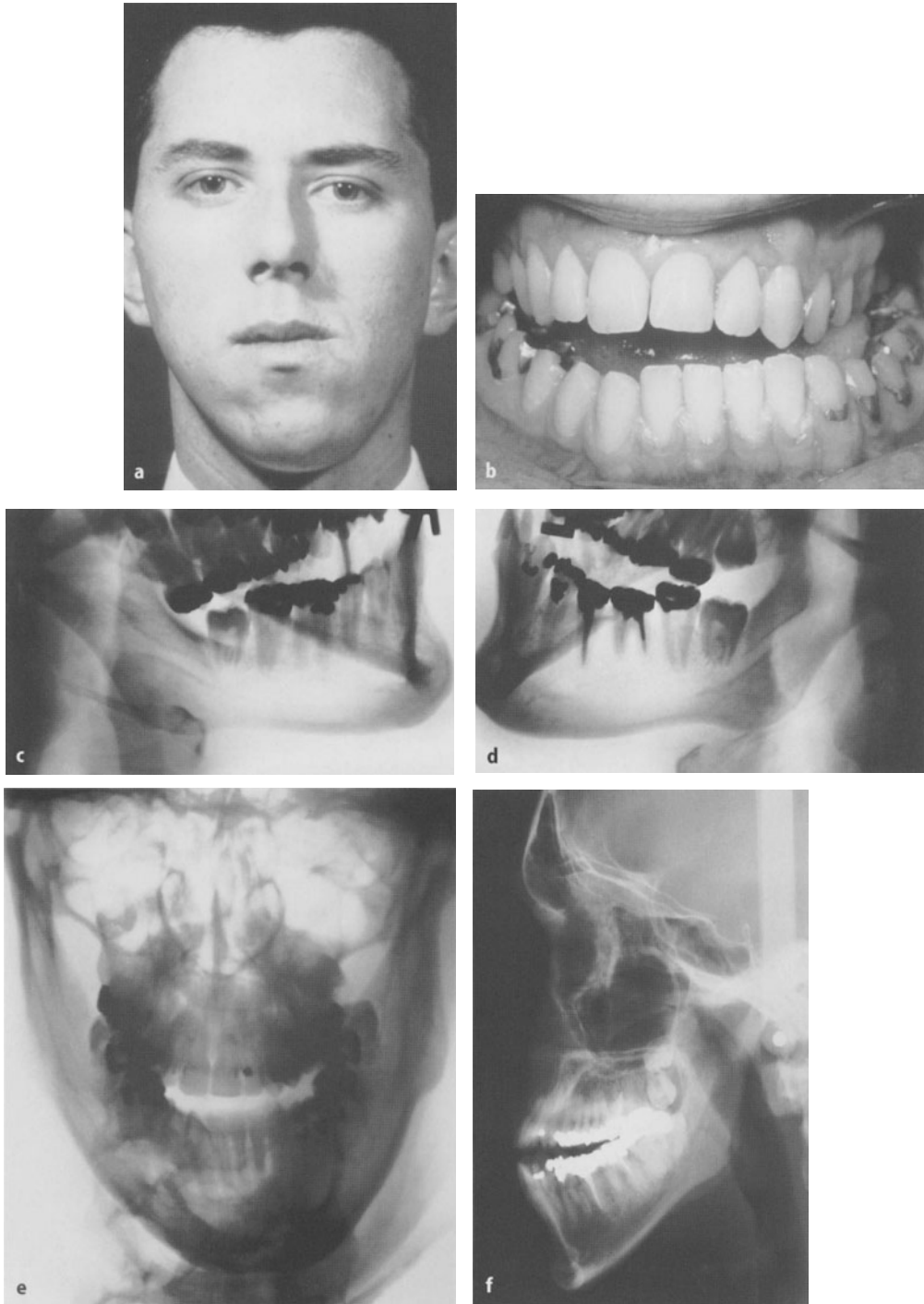


Fig. 57a–f. Bilateral H.E., slender type, producing a severe, asymmetrical open bite. **a, b** The patient's front view is like a mirror of his occlusal situation. **c, d** Right and left lateral oblique views of the horizontal and ascending rami allow the observer to easily recognize the shape of a bilateral H.E., slender type. **e** The p.a. projection of the mandible in the closed position shows the enormous difference in length of the two ascending rami with outward rotation of the angle on the shorter left side. **f** The preoperative lateral cephalogram discloses that both sides of the mandible lie in almost a straight line from the condyles to the chin region and a certain degree of retro-maxillism

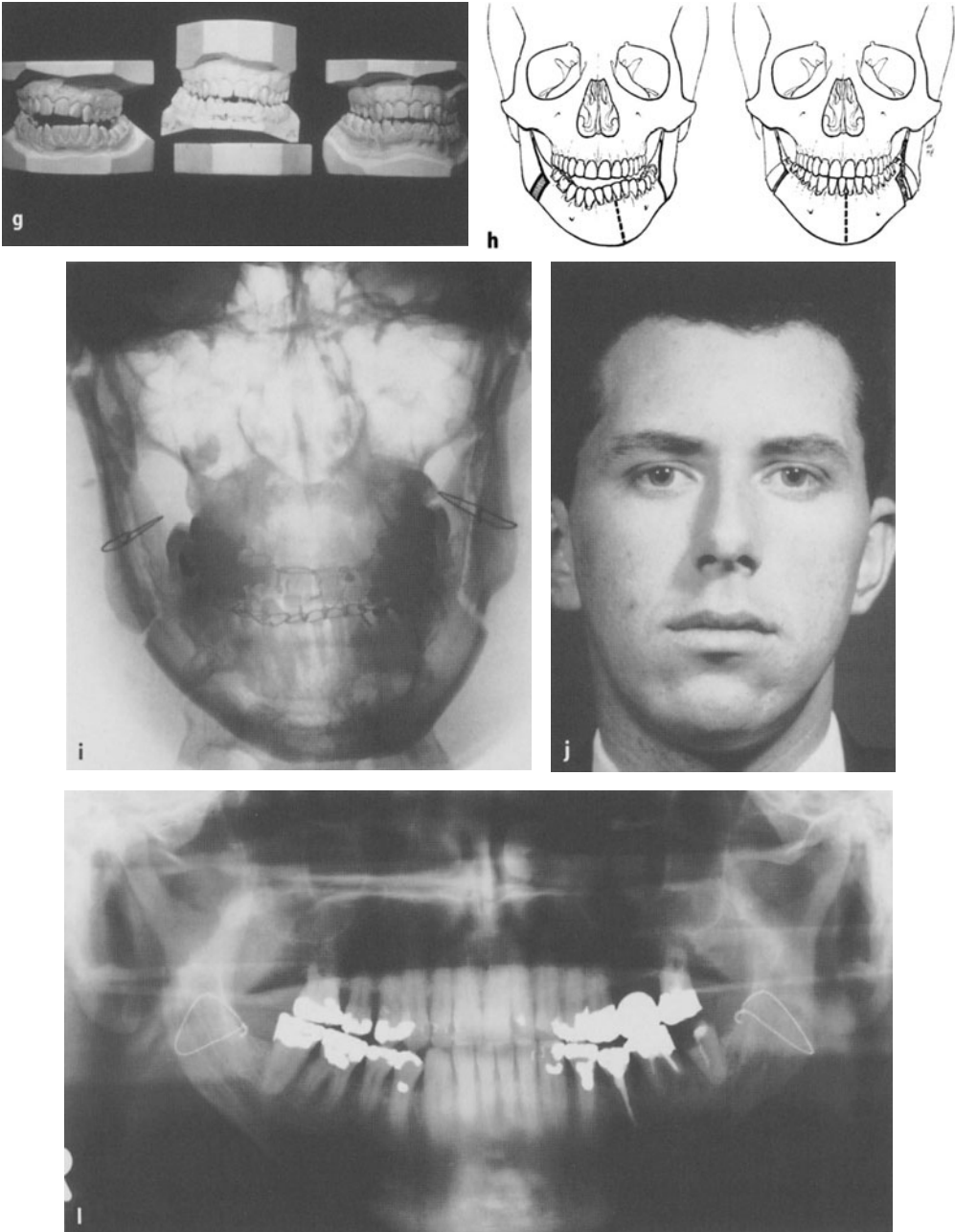


Fig. 57g–l. Bilateral H.E., slender type, producing a severe asymmetric open bite. **g** The model operation reveals that a very acceptable occlusion can be achieved by performing bilateral sagittal splitting of the rami for mandibular repositioning. **h** Schematic illustration of the bilateral rami sagittal splitting procedure producing the planned occlusion and appearance. **i** The postoperative p.a. radiograph of the mandible shows the long contacting bone surfaces securing early and firm bony union. **j, l** The panoramic view taken 22 years after surgery still permits the observer to recognize the former shape of a bilateral H.E., slender type, of the mandible

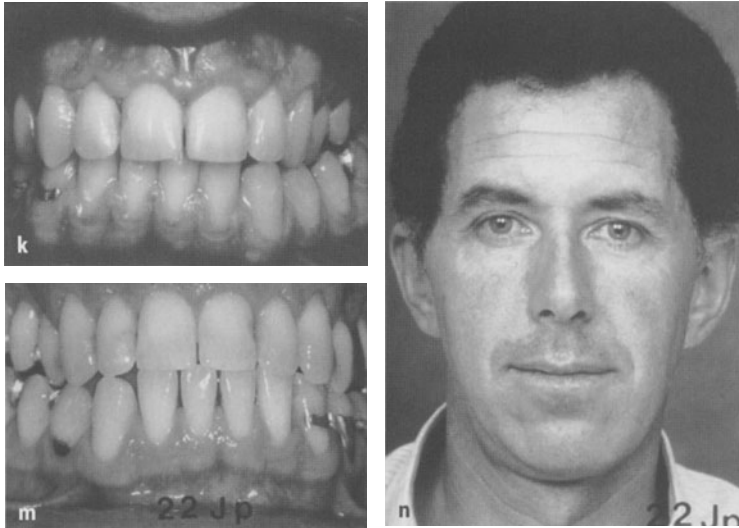


Fig. 57 **k, m, n.** Bilateral H.E., slender type, producing a severe asymmetric open bite. **k** The patient's appearance and occlusion 1 year and 2 months after surgery. **m, n** The patient's appearance and occlusion 22 years after surgery

Bilateral H.E., Unilateral Slender and Contralateral Non-Slender Type (Fig. 58)

Sex and Age. Male, 28 years of age.

Aetiology and History. The patient knew no reason why his mandible had grown forwards. He reported that his father also had a protruding mandible. He did not know whether or not it was also asymmetrical, as his father had died when he was a boy.

The forward growth of the mandible started when he was about 12 years old. It had not increased during the last few years.

Clinical Findings. Looking at the patient from in front (Fig. 58a), his very severe mandibular asymmetry with an antemandibulism appearance cannot be missed. The chin is rather broad and out of the facial midline by 12 mm to the right. The middle third of the face is flat. The lip commissure is nearly horizontal, but its right corner is further lateral than the left. The upper lip seems to be drooping on the right side. There is very little vermilion on that side. Mouth opening is unimpeded, but deviates to the shorter right side but less than in the closed position.

In profile view (Fig. 58b) from the left side, the very long mandibular contour without an angle at all presents a rather unusual picture. There is not a real antemandibulism profile as the lower lip is only very slightly in front of the upper lip, while the high chin is further back.

The teeth in the closed position (Fig. 58c–e) show a severe crossbite on the right side and in the front region, with a slight class III occlusal relationship in the molar region. On the left side there is a class III relationship by the width of more than two lower molars. The lower teeth midline is shifted over to the right side by $1\frac{1}{2}$ lower incisors width. Function and innervation of the facial soft tissues are undisturbed.

Radiographs. The panoramic view (Fig. 58f) shows a very much elongated left mandible with the elongated, slim condylar neck, ramus and body almost in a straight line. There is very little protuberance in the angle area, and the body height in the last molar region is only just enough to cover the roots of the third molar with bone. Its distal root extends down almost to the strong cortical plate. The right ascending ramus is at least of normal shape, but also slightly elongated. There is a certain angle protuberance though not really very much. The angle is also stretched and the body height below the roots of the molars is also less than normal, but not as much reduced as on the longer left side. The occlusal situation is the same as described above. The chin is separated into two prominences, the right stronger than the left. The right condyle of normal size, sits on a rather thick neck, the anterior surface of which seems some-

what elongated. A lower bicuspid is missing on the left side.

The TMJ-ramus tomograms (Fig. 58g, h) show, on the left side, the classic picture of a slender type H.E., while the right condyle is, in size and shape, within the normal range but with a thick and slightly elongated anterior neck surface.

The mandible in p.a. projection in the closed position (Fig. 58i) also shows clearly the enormous elongation of the left mandible with the more prominent right half of the cleft chin. The left horizontal, and even more the ascending ramus, look curved.

The lateral cephalogram (Fig. 58j) shows that the mandible protrudes beyond the maxilla by 2–3 mm.

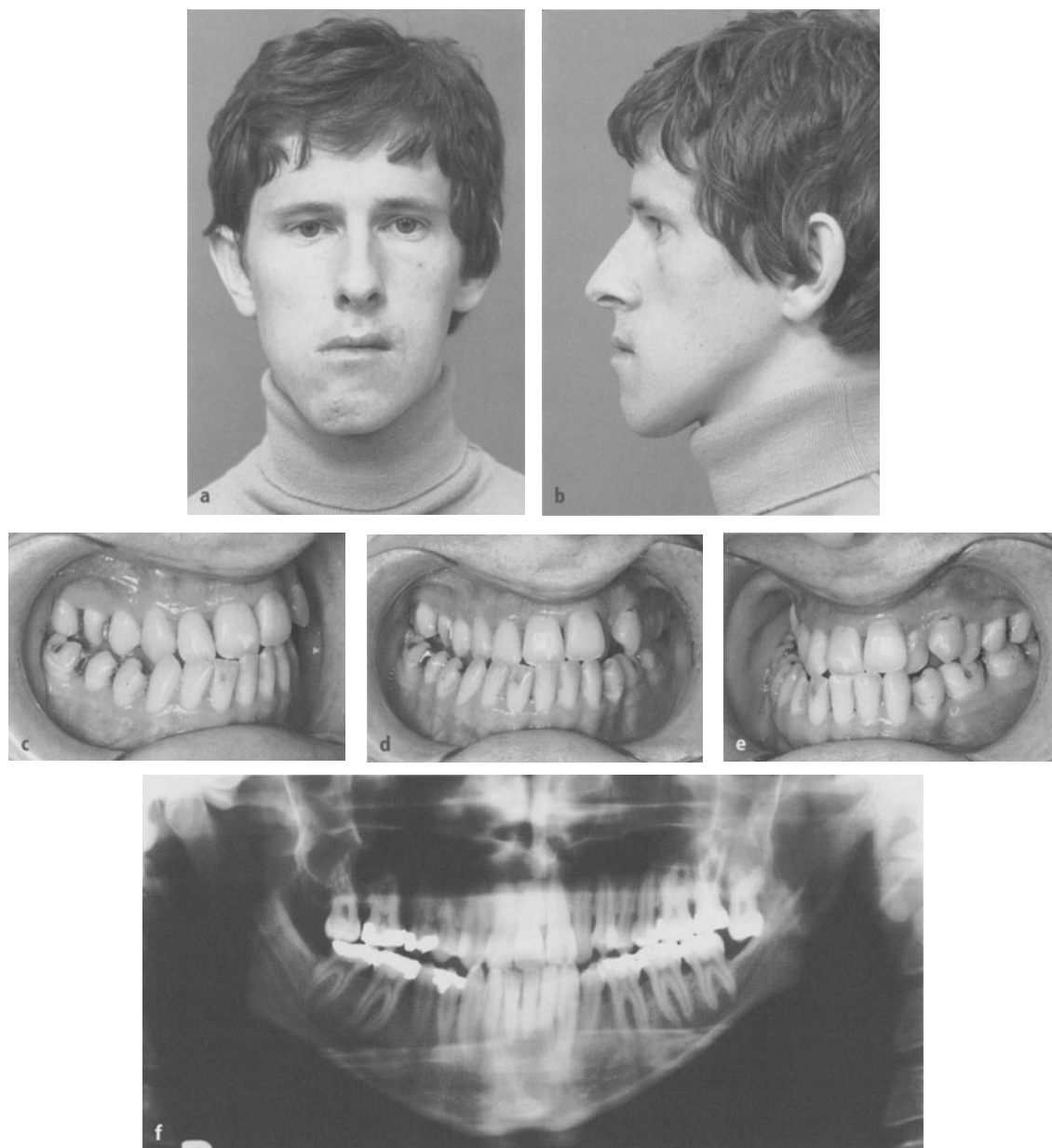


Fig. 58a–f. Bilateral H.E., unilateral slender and contralateral non-slender type. The patient's **a** front and **b** profile views show a very atypical antemandibulism (prognathic) facial appearance. **c–e** The patient's occlusion is that of an asymmetric antemandibulism. **f** The panoramic view discloses the background of this very severe mandibular deformity: an extreme H.E., slender type on the left side and also a clear H.E. non-slender type on the right side

The maxilla has a somewhat short base but is in an acceptable position. This projection also shows the difference in body height of the two sides of the mandible. Both lower borders are rather slightly concave. It might be worth while mentioning that the side which extends further down has no convexity of its lower border.

The tracing of the panoramic view (Fig. 58k) gives all the details of this bilateral asymmetric mandible (see Fig. 4 for abbreviations):

	R	L		R	L
HRL	118	120	APB	23	24
HRH 6/7	30	26	TL	63	54
HRH 3/4	42	43	WC	15	11
ARL	90	84	WN	14	10
APL	28	34	<	125°	143°

These measurements clearly mean that the total length of the right ascending and horizontal rami together measures 4 mm more than the left. The conclusion is that a lot of the elongation of the left side is due to the stretching of the angle.

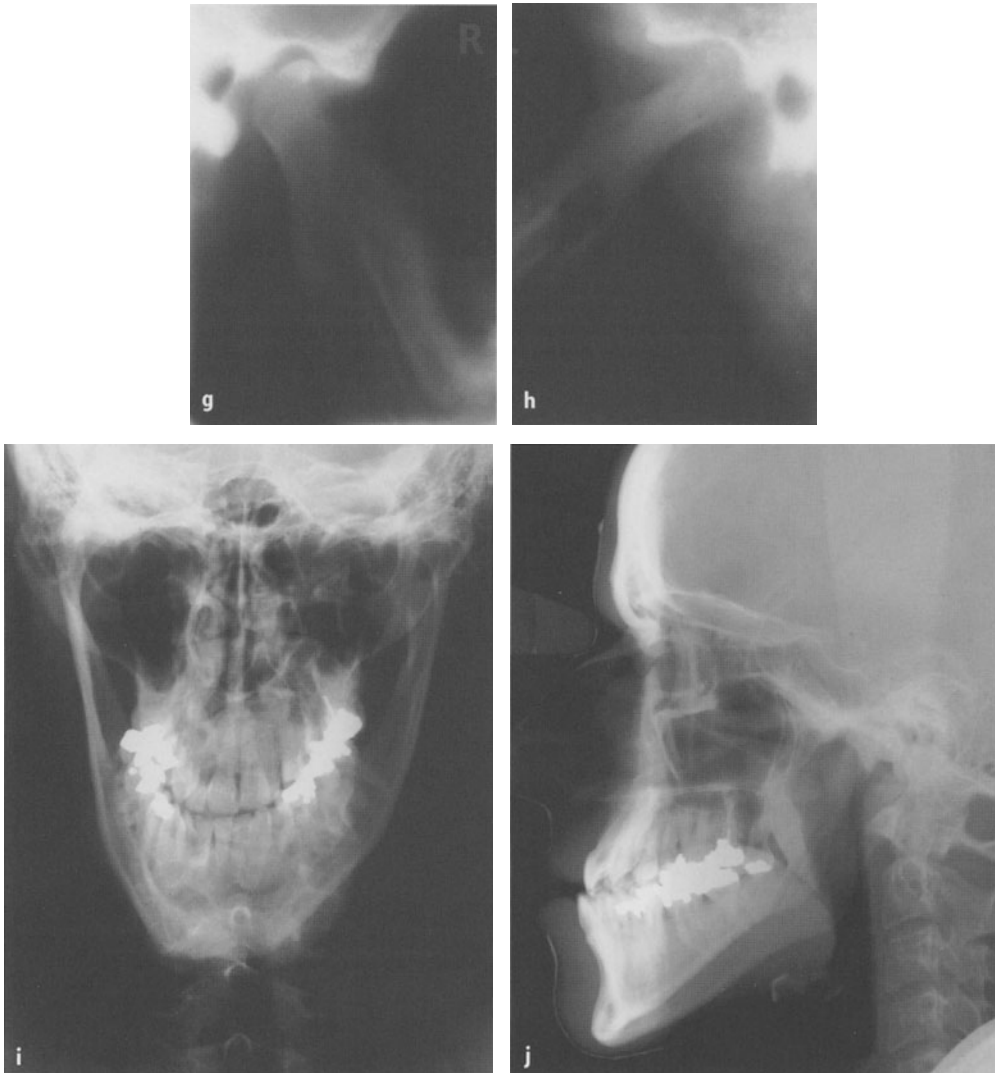


Fig. 58g–j. Bilateral H.E., unilateral slender and contralateral non-slender type. **g, h** The TMJ-ramus tomograms confirm what is seen on the panoramic view. **i** The mandible in p.a. projection in the closed position permits the clear diagnosis of an asymmetrical, bilateral H.E. **j** The lateral cephalogram shows the antemandibulism anomaly but not the real skeletal background of it

Provisional Diagnosis. Bilateral H.E., severe slender type on the left and less severe non-slender type on the right side.

Treatment Plan. Presurgical orthodontics followed by repositioning of the mandible by bilateral sagittal splitting of the rami.

Actual Treatment and Result. As the mandible could be brought into a fairly good occlusion according to the model operation, the patient refused to undergo presurgical orthodontics. The bilateral splitting of the rami permitted us to obtain the occlusion as planned in the model operation. The original splitting procedure was

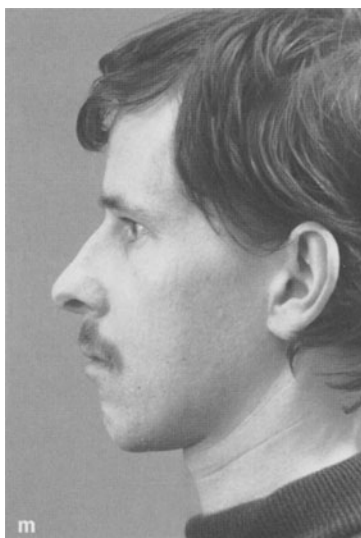
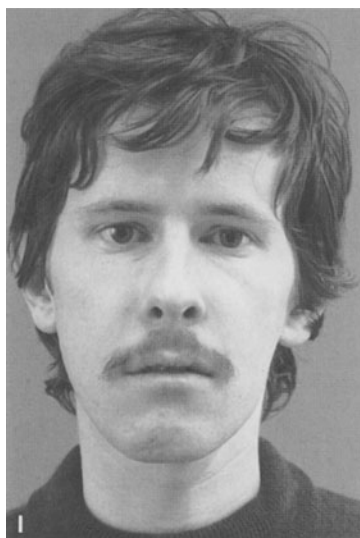
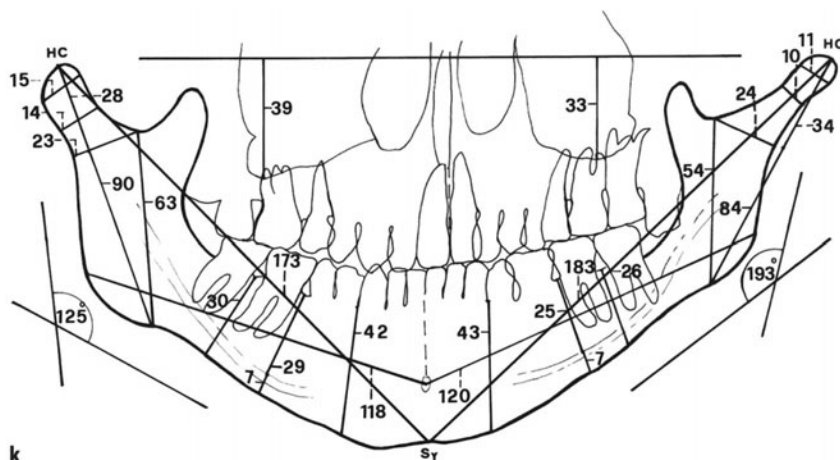


Fig. 58k–p. Bilateral H.E., unilateral slender and contralateral non-slender type. **k** The tracing of the panoramic radiograph and its measurements reveal very interesting facts about this bilateral mandibular anomaly. **l, m** Symmetry of the face and a normal mandibular contour was achieved. The patient had refused to have the chin improved in addition. **n–p** The patient's occlusion 1 year after correction, without any orthodontics

used in order to build up an acceptable angle area on the left side.

An anterior border wire and a circumferential wire and intermaxillary fixation for 6 weeks were used to ensure bone healing in the new position.

Clinically, a symmetrical face resulted (Fig. 58l, m) in a vertical profile line but with a still too high (deep) and retruded chin prominence. In the lateral view the length of the left mandible has been reduced substantially. The occlusion (Fig. 58n–p) still needs improvement by orthodontic treatment. It was refused by the patient.

I had suggested to the patient that I improve the appearance of his chin by moving its inferior rim forwards and upwards. He refused to have it done.

Discussion. This case, with a unilateral severe slender type of H.E. and less severe contralateral non-slender type raises the question: how was it that the H.E. anomaly was not the same type on both sides as it was in the case before this? The answer is simple: I do not know. I also do not know why an H.E. must not automatically be of the slender type. There are uni- and bilateral non-slender H.E. cases with almost just as much overgrowth in length as the slender type cases.

The fact, that the left slender type measured nearly the same length of the horizontal ramus as the right non-slender type is of special interest. The question, why

this is so, is simply answered. It all depends on the angle which the horizontal and the ascending rami form. If they form an almost straight angle (160° or even 180°) the total visible length would be the sum of the length of the ascending ramus plus the length of the horizontal ramus. If they form an almost right angle (100° or even 90°) then the total apparent length would be that of the horizontal ramus only. So, it is not surprising that in this case the sum of the right horizontal and of the ascending rami together was larger than that of the left slender side, although the slender side appeared clearly longer than the non-slender side and measured actually 10 mm more between the Sy-point and the HC-point.

Conclusion. The combination of an H.E. anomaly on one side with the typical severe slender form, and the contralateral side with the less severe non-slender type, is rather rare, but it can happen. The occlusion will be the same as in cases in which the same type of anomaly occurs bilaterally. One would expect that there must be a clear difference in the angle regions looking at the patient from the side. But experience in acromegaly cases proves that the shape of the skeletal abnormality does not automatically produce a corresponding outward appearance (see Fig. 76). Of special interest is the fact that the size of the angle makes all the difference whether a mandible appears long or not.

Bilateral H.E., Slender/Non-Slender Type in a Case of Normomaxillism (Fig. 59)

Sex and Age. Male, 21 years of age.

Aetiology and History. The patient was referred for correction of his occlusal anomaly because of pain in his TMJ areas. When he was a child he had had orthodontic treatment for several years. He did not know when his jaw anomaly was first noticed or by what it was caused. Nothing similar existed in his family history.

Clinical Findings. Looking at the patient from in front (Fig. 59a), a mandibular asymmetry is obvious, with the chin deviated to the left side. The left corner of the mouth seems more lateral than the right. The lip commissure is almost horizontal, slightly higher on the left side. The lower third of the face is too long. Looking at the patient from the side (Fig. 59b), he has a slight antemandibulism profile with the lower lip a little in front of the upper and a shallow labiomental fold. Musculature and facial nerves were intact. The oral aperture is normal in width, and the asymmetry is hardly detectable (Fig. 59c). The right TMJ was painful on palpation. There was clicking in both joints.

On closing the teeth, a severe occlusal asymmetry with an open bite is visible in the left anterior region, caused by a kink in the canine-incisor region of the lower dental arch (Fig. 59d–f). Both sides are in a class III relationship. The upper teeth are in a vertical position, but there is tilting of the lower teeth lingually and to the longer right side.

Radiographs. The panoramic view of the jaws in the closed position (Fig. 59g) shows a full dentition with the symphysis region and the midline of the teeth of the mandible over to the left by the width of almost one lower incisor. It also shows the bilateral class III molar relationship. Both condyles are well formed and of rather large size with wide necks, the right longer and wider than the left. Its condyle is a bit oversized. The lengths of the ascending rami are unequal but within normal limits for that size of mandible. The right angle is slightly stretched and the body height in that molar area is reduced compared with the left side. The right

body is longer than the left and longer than normal. The left body is almost normal in length and height, but its angle is also slightly stretched. The left ascending ramus seems shorter than the right one, but rather on the

long side because of the thick and elongated condylar neck. There is an open bite from the right upper canine to the left upper bicuspids. The trabeculation is unobtrusive.

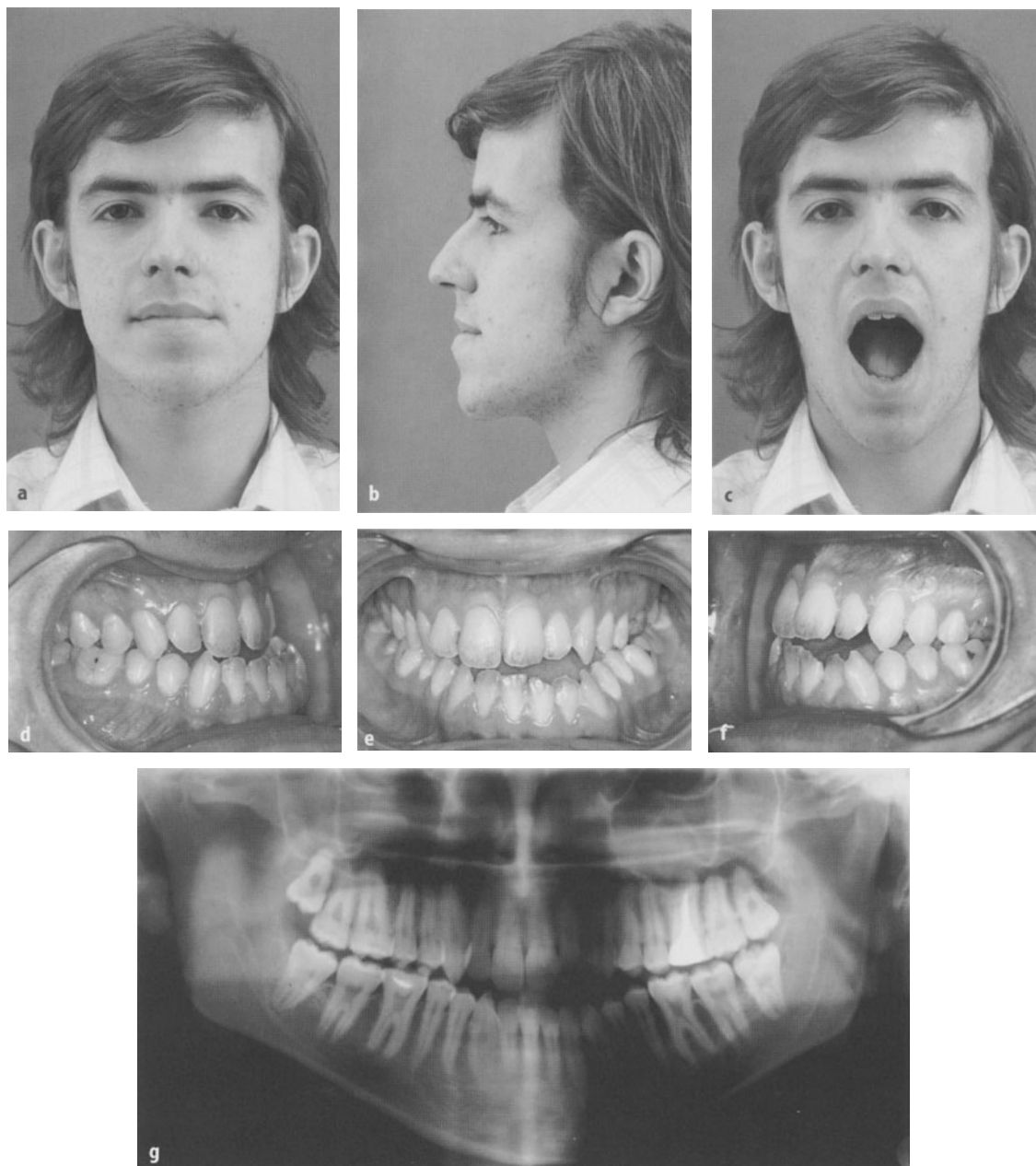


Fig. 59a–g. Bilateral H.E., slender/non-slender type, and normomaxillism. **a–c** The patient's front view shows the typical appearance of a so-called asymmetric prognathism. In profile view the face also appears compatible with that term. At maximum mouth opening there is still some deviation to the left side, but less than in the closed mouth position. **d–f** The patient's occlusion shows a bilateral class III relationship with a shift of the lower teeth midline to the left by the width of the lower incisor and there is an open bite in the left incisor-canine region with a clear kink there. **g** The panoramic view shows nothing spectacular in the shape of the mandible, but there are some differences between left and right sides

The preoperative TMJ–ramus tomograms were of rather poor quality. Since the condyles and their necks do not change shape or size following surgery, I take the

liberty of using those taken a few days after surgery (Fig. 59h,i). Both sides show more clearly what had been seen in the panoramic picture: the right neck is clearly

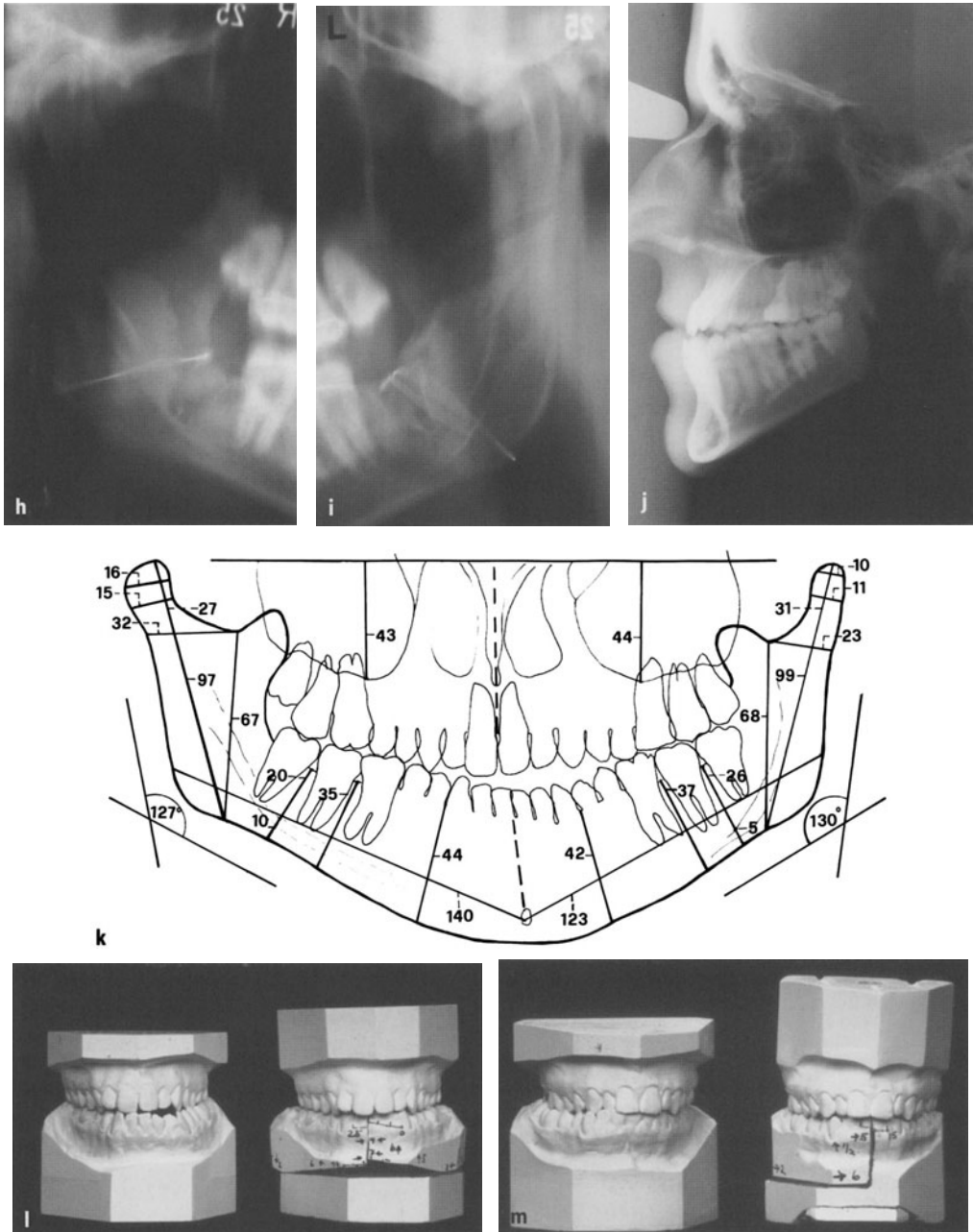


Fig. 59h–m. Bilateral H.E., slender/non-slender type, and normomaxillism. **h, i** The TMJ–ramus tomograms show the condyle and neck situation of the two sides quite clearly. **j** The lateral cephalogram shows a minor degree of antemandibulism due to a long and strong mandible with a normomaxillism situation. **k** The tracing of the panoramic view and its measurements disclose astonishing differences between the left and right hemimandibles. **l** The model operation intends to reduce the excess length of the right hemimandible by a parasymphysiotomy associated with the extraction of tooth 41. With an additional bilateral sagittal splitting of the rami and mandibular repositioning, a very acceptable occlusion will be achievable. **m** In unilateral cases of H.E., parasymphysiotomy without sagittal ramus splitting may be all that is needed

thicker and longer than normal and its head is a bit more voluminous than the neck. The left side shows a slimmer condyle and neck which clearly widens to-

wards the semilunar notch. But it is also elongated compared with the normal length.

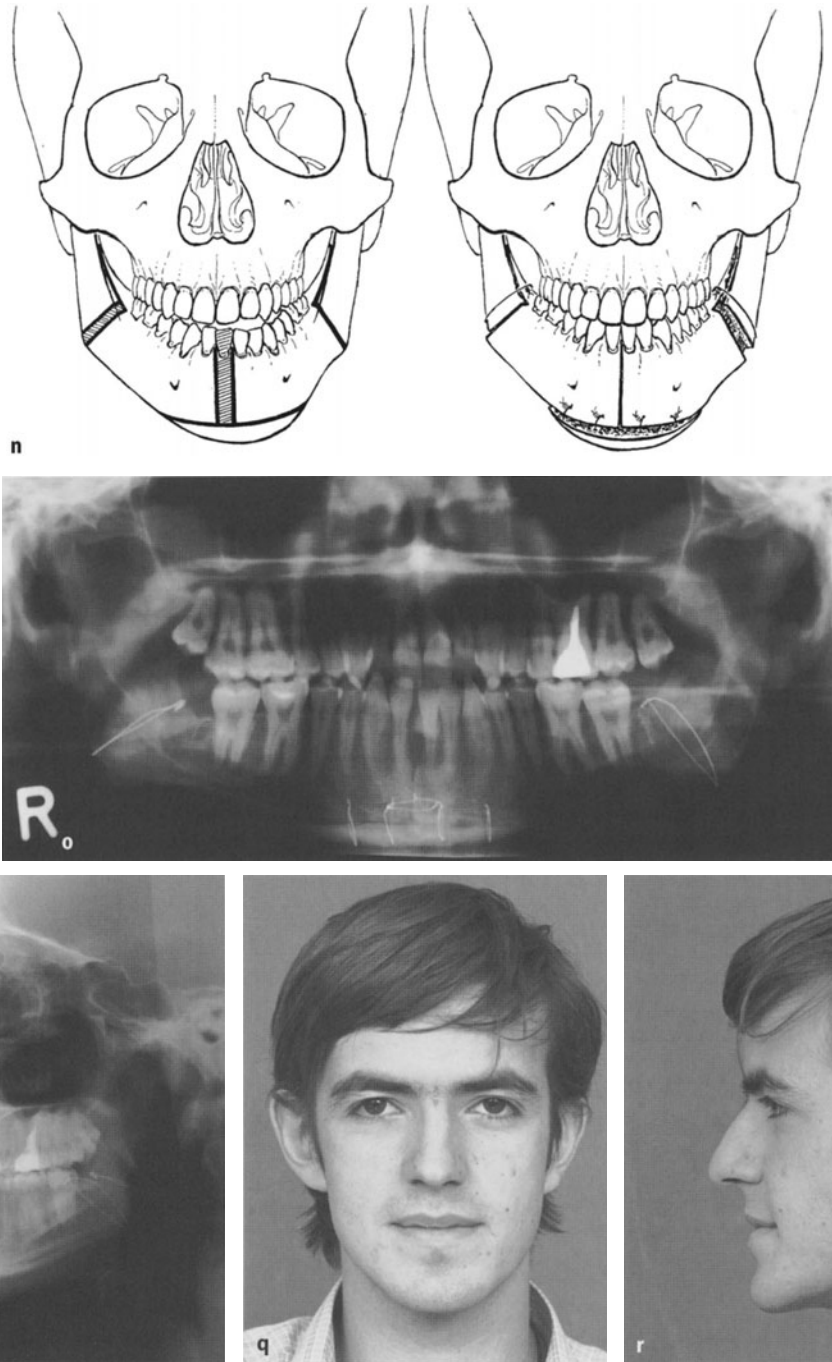


Fig. 59n-r. Bilateral H.E., slender/non-slender type, and normomaxillism. **n** Diagrammatic illustration of the planned surgery. **o** The panoramic radiograph, taken 8 weeks after surgery, permits visualization of the various osteotomy lines. **p** The lateral cephalogram taken 1 year after correction shows the final skeletal relationship. **q, r** The patient's appearance and profile views 1 year after correction

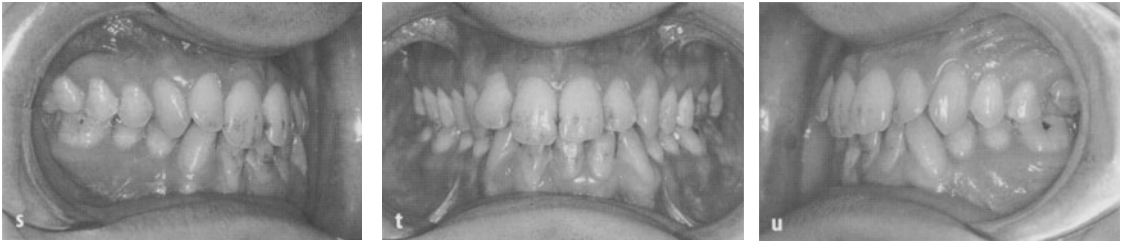


Fig. 59s–u. Bilateral H.E., slender/non-slender type, and normomaxillism. **s–u** The patient's very satisfactory occlusal situation 1 year after surgery. No postoperative orthodontics was undertaken

The mandible in *p.a.* projection with the mouth open did not show anything remarkable, only that the whole mandible seemed fairly symmetrical and rather strong.

The lateral cephalogram (Fig. 59j) reveals the slight class III profile and the open bite with a very long mandible of normal shape. The maxilla is normal in size and position (normomaxillism).

The tracing of the panoramic view (Fig. 59k) gives all details regarding differences between the right and left sides. Although the two halves seem to look almost alike on the radiograph, measurement discloses the truth: the ascending rami are almost equal in total length, but differ remarkably in their trunk lengths and the articular processes. The main difference is found between the length of the two horizontal rami, the right being 17 mm longer than the left. Both angles are within the normal range.

Provisional Diagnosis. Bilateral H.E., slender/non-slender type with an open bite. The left side closer to the slender type.

Treatment Plan. According to the model operation (Fig. 59l) the best result would be achieved by mandibular repositioning with a bilateral sagittal splitting procedure on the ascending rami, para-symphysiotomy including the right lower first incisor and a chin rim advancement with lateralization to the right. The graphic illustration shows clearly the type of corrective surgery planned (Fig. 59n).

Actual Treatment and Result. The surgery was carried out as planned, in routine fashion, using the bilateral sagittal splitting procedure with circumferential wires round the rami and direct chin wires, with 6 weeks intermaxillary fixation. The panoramic view, taken just after removal of the intermaxillary fixation wires, permits still clear visualization of the various osteotomy lines (Fig. 59o). The final result including the occlusion as well as the patient's appearance turned out as planned (Fig. 59p–u).

Discussion. This is a classic case of unequal bilateral H.E. of a slender/non-slender type on the right side and a nearly pure slender type on the left side in a rather strong mandible. Diagnosis and surgery did not cause any problems.

Since we believe in the existence of the two M and L condylar growth regulators we also understand the asymmetrical hyperactivity of the same growth regulator on the two sides. We do not know how they are activated. This case shows clearly that there can be differing degrees of hyperactivity and that they may also produce differing shapes and lengths of the neck of the condyle.

In the H.E. cases it is often worthwhile, by the model operation, to try to see whether the extraction of the first lower incisor on the elongated side with concurrent parasymphysiotomy can produce a better occlusal result than without it. This is particularly indicated when the patient cannot receive adequate presurgical orthodontics. I found that is rather often the case. In addition, it has the advantage that the longer side is shortened in the chin area. It always requires the detachment of the chin prominence as a muscle-pedicated bone flap. When a chin symmetrization is indicated it makes the decision easier.

When the H.E. is unilateral only, the parasymphysiotomy on the elongated side, including the first incisor, may be all that is needed to restore a good occlusion (Fig. 59m) and for correction of the mandibular asymmetry.

Conclusion. A bilateral H.E. can have different manifestations in the two sides of the mandible. The more active side overrides the other side. If one side is particularly active, then the mandible is not only pushed over to the opposite side but also downwards on the contralateral side producing there an open bite. Whether such a case primarily starts from a normomandibulism genetic pattern or from an antemandibulism genetic pattern we do not know. In planning the surgical correction, the extraction of the first lower incisor on the longer side followed by a corresponding symphysiotomy is often worth trying.

Bilateral H.E. Slender Type and Micro-Retromaxillism (Fig. 60)

Sex and Age. Male, 34 years of age.

Aetiology and History. The patient had no idea why his lower jaw had started to grow forwards. It was already noticeable when he was aged 8. There was no similar case in his family history.

Clinical Findings. This very tall slim patient has a rather long slim face (Fig. 60a). The middle third is flat (dish-face deformity). The nose inclines to the left side. The mandible protrudes severely, with a shift to the right side of about 11 mm. The shape of the mandible is almost triangular with a horizontal chin prominence. The gonial angles are clearly visible and widely open (Fig. 60b). Mouth opening was normal.

The teeth are in a rather poor condition (Fig. 60c–e). The lower front teeth are clearly retruded with their midline over to the right side by more than one lower incisor. There is a severe negative overjet of 17 mm. Only the last lower molars are in occlusal contact with the upper bicusps. The facial musculature was rather thin, the facial innervation was perfect.

Radiographs. The panoramic view (Fig. 60f) discloses two very long, almost straight hemimandibles, the left longer than the right. The abnormal length seems to be mainly due to an increase in the ascending ramus length. The condyles are of almost normal size and shape. The condylar necks do not seem to be especially elongated. They are almost in a straight line with the posterior borders of the ascending rami. There is not much of a mandibular angle, on either side. The angle of the shorter right mandible seems more stretched than on the left. Both mandibular bodies have very little height, particularly in the molar regions. The trabecular structure is inconspicuous.

The tracing of the panoramic radiograph (Fig. 60g) discloses the differences between the two sides. It is very interesting that the shorter right side has a shorter ascending ramus (89 mm) than the left (91 mm) despite a 3 mm longer articular process, together with a larger gonial angle (142°) than the left side (136°). That shorter articular process on the left side is well compensated by a 6 mm longer trunk of that ascending ramus. It is also worth mentioning that the height of the horizontal ramus in the molar region is 10 mm less on the left, longer side. The main length difference seems to be between the two horizontal rami (8 mm).

The TMJ–ramus tomograms (Fig. 60h, i) show rather smaller condyles than normal with very long and slim necks, the right even a little longer than the left, contrary to what they looked like in the panoramic view. This proves that the panoramic view may not demonstrate the real situation.

The mandibular p.a. projection in maximum mouth opening (Fig. 60j) shows nothing remarkable, other than the extremely long hemimandibles with very fine trabeculation, and a rather broad chin.

The lateral cephalogram (Fig. 60k) discloses the tremendous discrepancy between the mandible and the maxilla. The very long, nearly straight mandible lacks normal height and protrudes over the maxillary teeth by 18 mm. The base of the maxilla is rather short and severely retrodisplaced (micro-retromaxillism).

Provisional Diagnosis. Bilateral, asymmetrical, very impressive H.E., slender type and micro-retromaxillism.

Treatment Plan. The planning was done as usual (for details see case Fig. 63). In this case it emerged that the mandible had to be repositioned posteriorly by 15 mm and the maxilla be brought forward by 10 mm. These measurements were the basis of performing the model operation. For that, a pair of models were fixed in the articulator in accordance with the bite registration.

The model operation disclosed that a very acceptable occlusion could be achieved, if in addition to the reverse movement of the maxilla and the mandible by the amounts indicated on the planning sheet, a rotation of the mandible to the left by 8 mm were added (Fig. 60l).

For the Le Fort-I osteotomy advancement, a bone graft from the iliac crest was required to bridge the rather large gap in the canine fossa areas and to increase the contour of the flat middle third. In addition, the sagittal splitting procedure on the two rami was to be done with the lateral cortical cuts directed to the angle area, to provide the possibility of improving the angles (Fig. 60m).

Actual Treatment. The surgery was carried out as planned, using wires for the stabilization of the maxillary advancement and circumferential wiring of the rami and 6 weeks intermaxillary fixation. The planned result was achieved. On the lateral cephalogram the gonial angle has changed from 141° to 132° (Fig. 60n). The patient's appearance and occlusion became very satisfactory (Fig. 60o–s). The patient refused to have the obliquity of his nose corrected also.

Discussion and Conclusion. Bilateral H.E., in particular the slender type, can produce an excessively long lower jaw. The elongation is only partially due to the elongated condylar neck, but it is also due to an increase in the ascending ramus height and to the stretching of the angles of the neck-ramus region and the mandibular an-

gles as well, both producing additional lengthening of the mandible.

The panoramic view rather often does not show the real anatomy of the TMJ–ramus situation. Only additional tomograms will reveal this clearly.

With regard to the final vertical profile, I personally prefer the straight anteproof type for males and fe-

males whenever possible. To achieve a straight anteproof line in this case, would have meant the need to move the maxilla even further forward. That again would have required a Le Fort III and I mid-facial advancement, a much bigger operation.

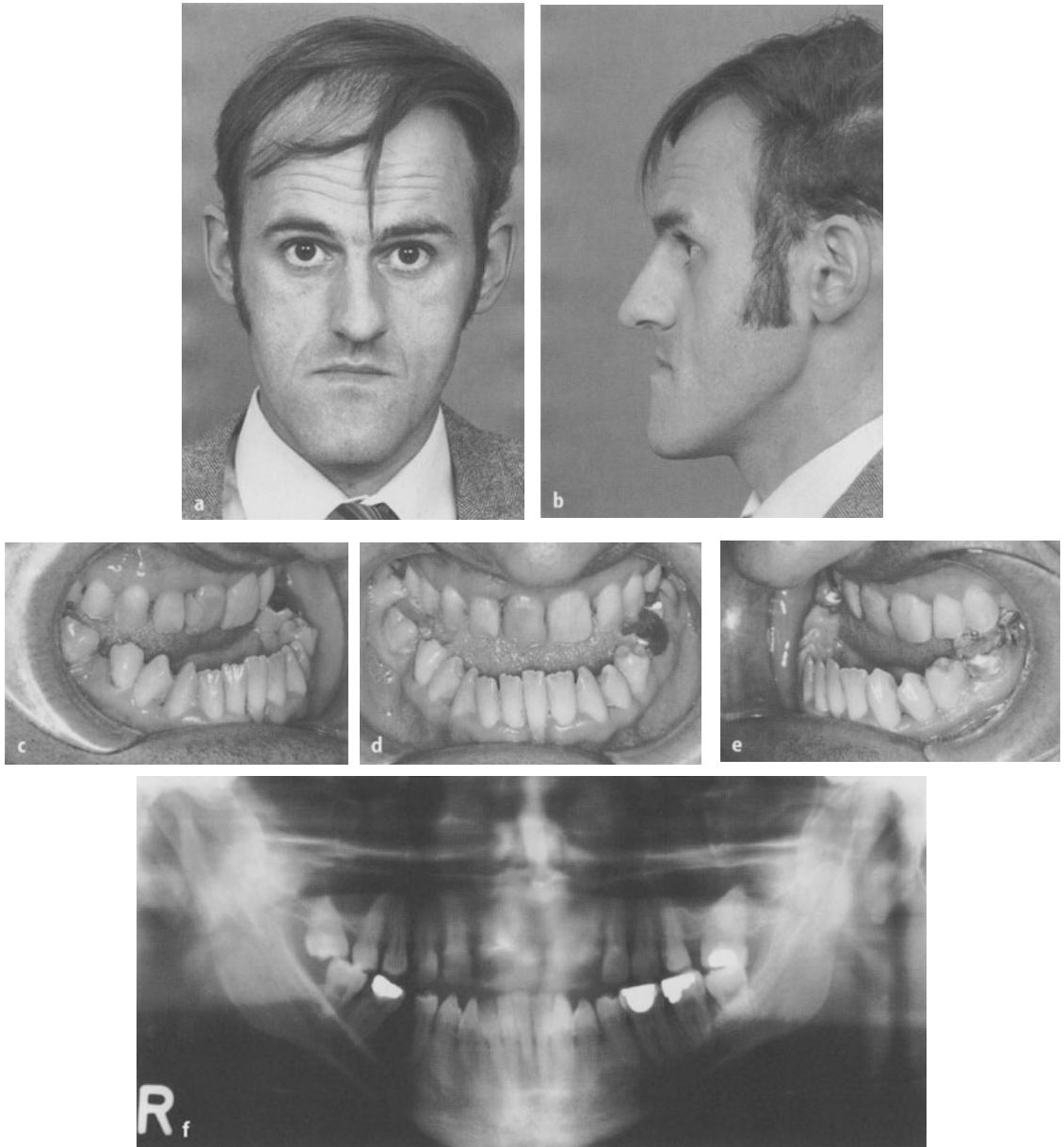


Fig. 60a–f. Bilateral H.E., slender type and micro-retromaxillism. The patient's **a** front and **b** profile views with a long asymmetrical protruding mandible and a very flat middle third. **c–e** The patient's occlusion with lingually tilted lower teeth and a midline shift to the right by one lower incisor. **f** The panoramic view discloses a very slim, long asymmetric mandible

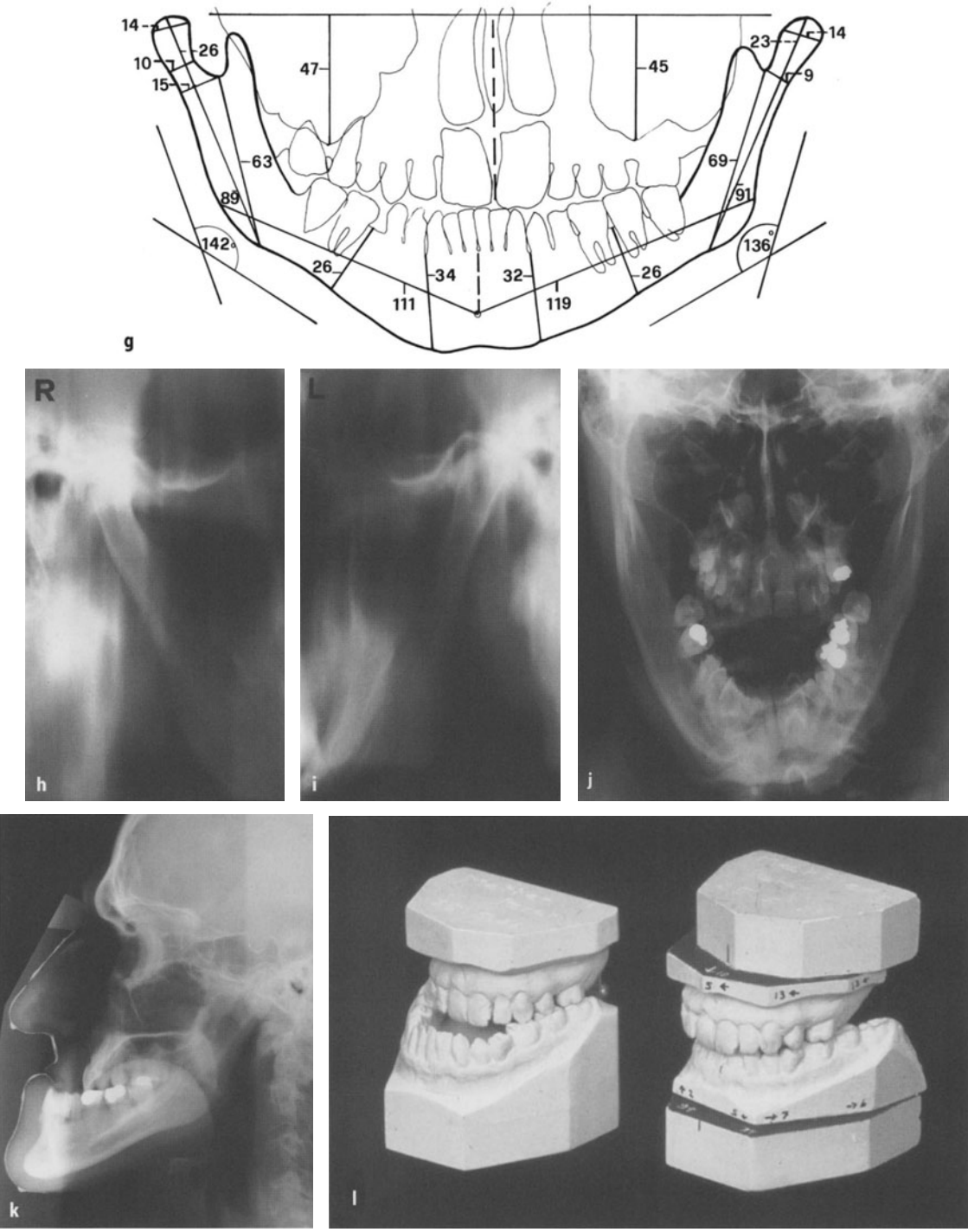


Fig. 60g-l

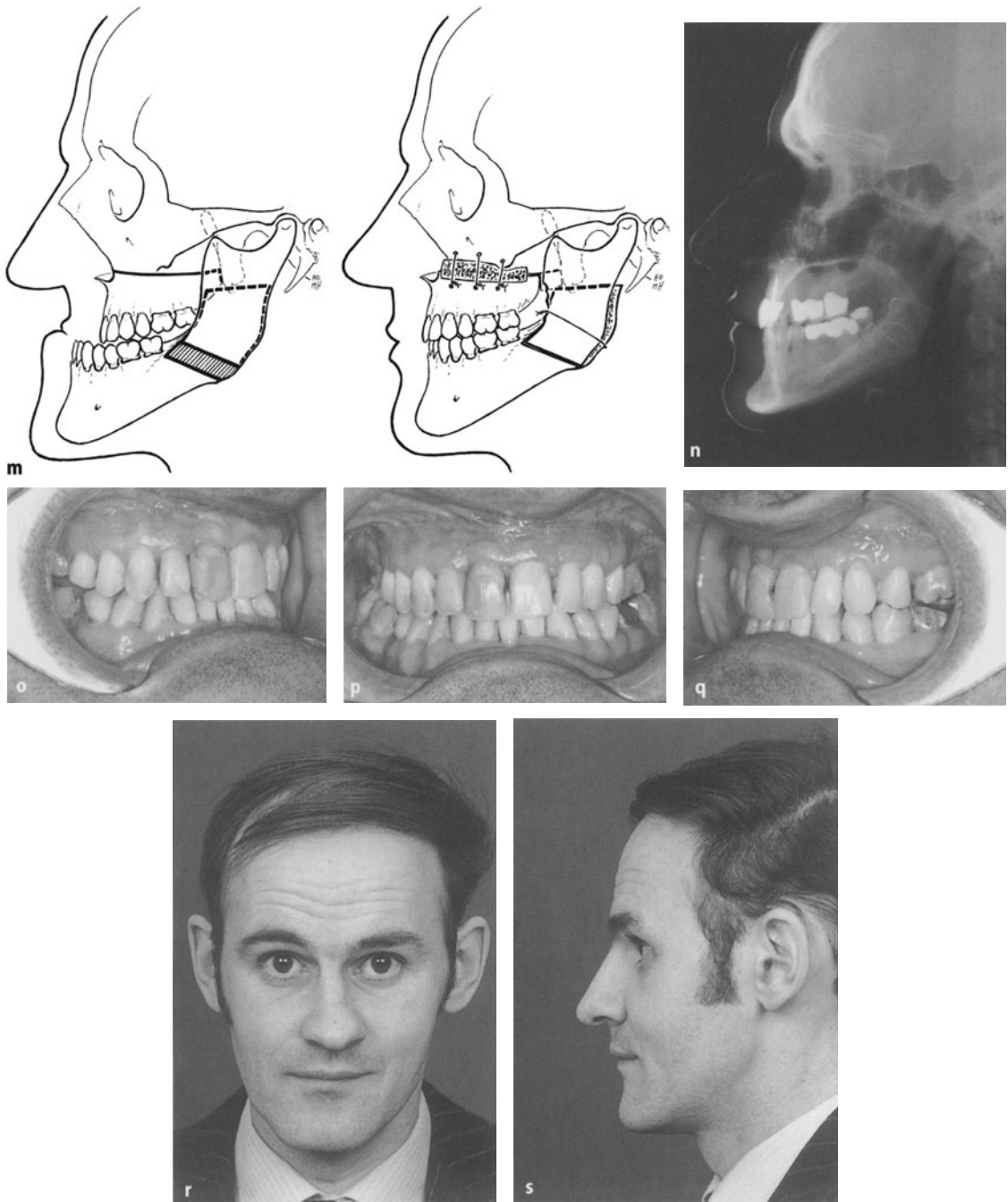


Fig. 60g–s. Bilateral H.E., slender type and micro-retromaxillism. **g** The measurements of the tracing of the panoramic view disclose the anatomical background to the differences in length of the two hemimandibles. **h, i** The TMJ–ramus tomograms disclose a better picture of the condyle–neck–ramus situation than the panoramic view. **j** The mandible p.a. projection in the open mouth position gives no additional information in this case. **k** The lateral cephalogram discloses the skeletal background to the maxillo–mandibular discrepancy very clearly: a long stretched mandible and a short retrodisplaced maxilla. **l** Model operation: moving the maxilla and the mandible according to the planning measurements should create a good occlusion. **m** Diagrammatic illustration of the planned operation. **n** Lateral cephalogram taken 1 year after surgery. The angle area has changed from 141° to 132° . **o–q** Occlusion 1 year after correction. **r, s** The patient's final appearance, 1 year postoperatively

Bilateral H.E., Slender Type, with Circular Open Bite in a Case with Medium Degree of Micro-Retromaxillism (Fig. 61)

Sex and Age. Female, 22 years of age.

Aetiology and History. The abnormal forward growth of the mandible was first noticed at the age of 8 years. It had gradually become more prominent and had remained stable since the age of 17 years. The patient came for surgical correction at the age of 21 years. The patient knew nothing which could have been a possible cause, and there was nothing of note in the family history.

Clinical Findings. The patient has an asymmetrical long face (Fig. 61a,b). The undue length is mainly in the lower third but partially also in the rather flat middle third. The mandible seems straight, from the ears to the chin, the right side longer than the left, with almost no angle. The lip commissure is horizontal, the left corner slightly further back than the right. The chin is over to

the left by 7 mm. Skin, musculature and facial innervation were normal. Mouth opening was normal.

All teeth are present except for three second bicuspid. Where they are missing, the deciduous molars are still present. The occlusion (Fig. 61c–e) shows a class III relationship in the molar regions and an open bite from molars to molars. All lower teeth are tilted lingually, particularly the incisors. In spite of this there is a negative overjet of a few millimetres. The lower teeth midline is over to the left by almost the width of a lower incisor.

Radiographs. The panoramic view (Fig. 61f) in the closed position shows a rather gracile mandible with obtuse angles and condyles of normal size but with elongated necks which make the usual open angulation with the posterior border of the ascending rami. The

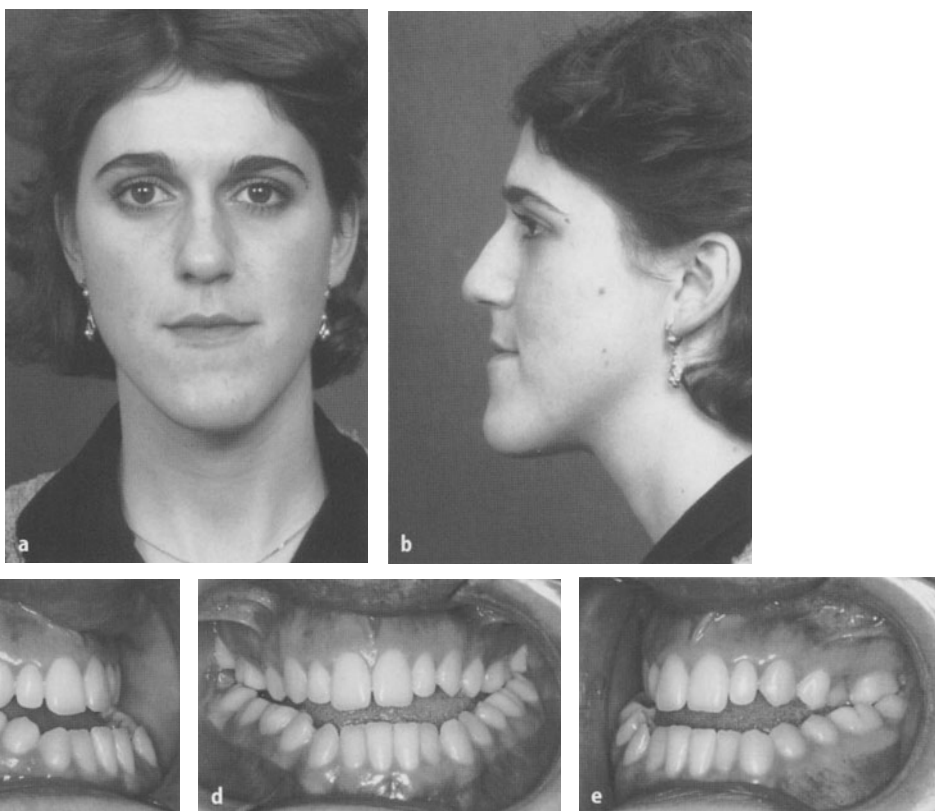


Fig. 61a–e. Bilateral H.E., slender/non-slender type with circular open bite in a case of medium degree micro-retromaxillism. The patient's **a** front and **b** profile appearance with a typical mandibular long face, an asymmetric antemandibulism and a medium degree flat middle third. **c–e** The patient's occlusion pictures show the asymmetric antemandibulism with lingual tilting of all teeth

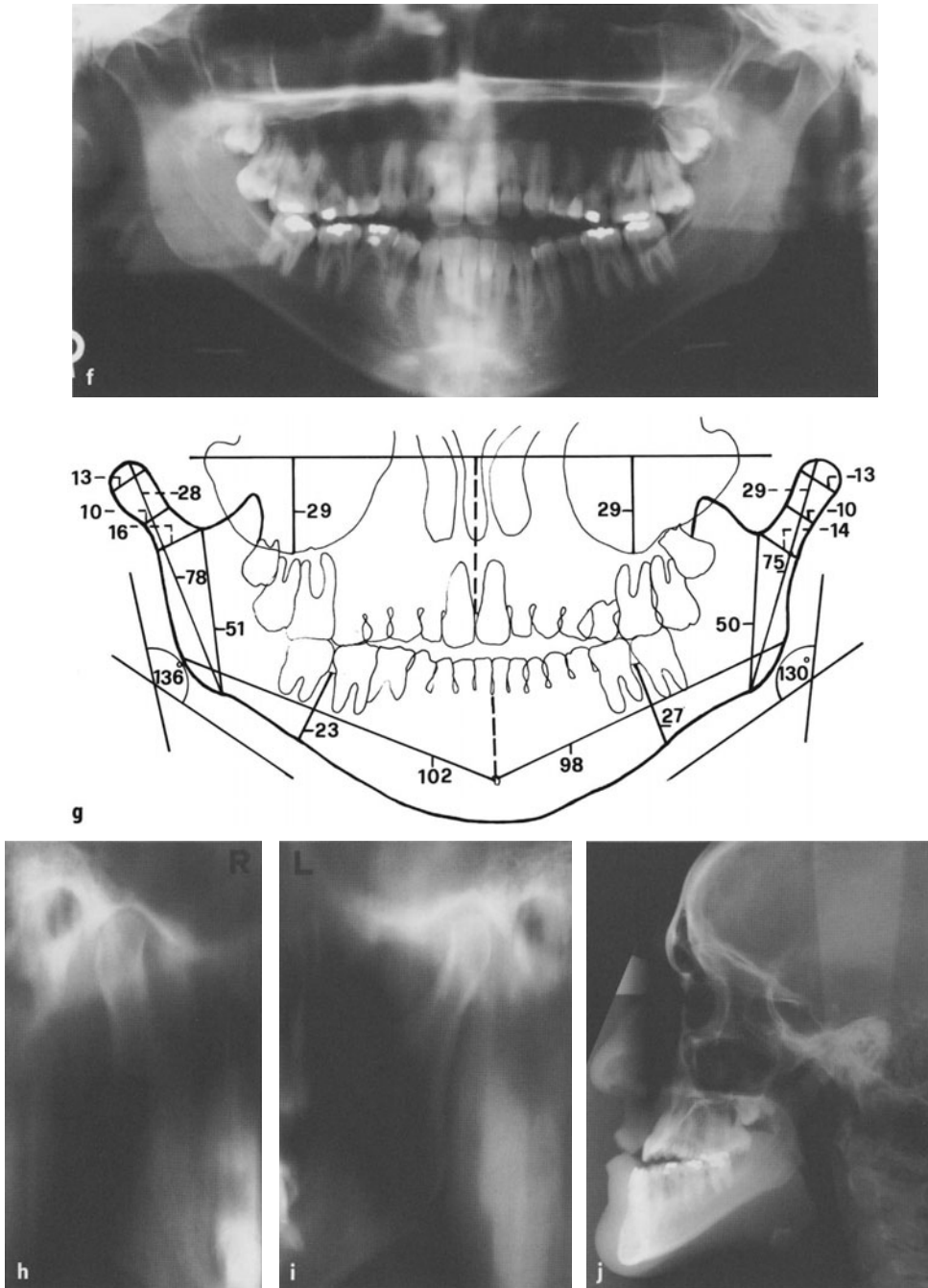


Fig. 61f–j. Bilateral H.E., slender/non-slender type with circular open bite in a case of medium degree of micro-retromaxillism. **f, g** The panoramic view permits the diagnosis of a bilateral medium degree slender type H.E. The measurements of its tracing do not disclose great differences between left and right sides, but all length measurements favour the right side. **h, i** The TMJ–ramus tomograms change that diagnosis as far as the ascending rami with the articular processes are concerned. **j** The lateral cephalogram discloses the long mandible with its retruded front teeth and the crowding in the maxilla because of its short base, which in addition was too retroposed

right side of the mandible seems slightly longer than the left. Although there is a clear lack of height in the mandibular body in the last molar regions, it is not so extensive as in the very severe H.E. slender type. The lack of height in the longer right side of the mandible is more obvious than on the other shorter side. Altogether it is the picture of a medium degree slender type H.E.

The measurement of the tracing of the panoramic radiograph (Fig. 61g) reveals almost equal values for the two hemimandibles. The right side shows the classic signs of the slender type H.E. more than does the left. This is true for the length of the horizontal and ascending rami and the gonial angle but not for the length and shape of the articular process. In that respect the left side is greater in its shape and length. All other length measurements are in favour of the right side H.E. anomaly and so is the vertical height of the horizontal ramus in the molar region, which is 4 mm less than on the left side.

The TMJ-ramus tomograms (Fig. 61h,i) disclose the real situation in the condyle-ramus area. Both condyles are within normal size limits, although tending to be on the smallish side, and both necks are very long and slim

but not thin. The ascending rami themselves are very short.

The lateral cephalogram (Fig. 61j) reveals that there is an almost straight mandible, quite protrusive with its retruded incisors projecting just in front of the upper front teeth, with a micro-retromaxillism of medium degree.

The lateral cephalogram after presurgical orthodontics (Fig. 61k) discloses the actual situation of the jaws and their teeth before surgery.

Provisional Diagnosis. Bilateral asymmetric H.E. slender type with circular open bite and severe lingual tilting of all lower teeth, in a case of micro-retromaxillism of medium degree.

Treatment Plan. As follows:

1. Presurgical orthodontics: this has to adhere to several principles (Principle No. 25). In this case very important is: a.) "Do not leave more teeth in the alveolus than can be placed in proper angulation to the respective jaw base". This will entail extraction of one tooth on each side of the maxilla because of its micro-ret-

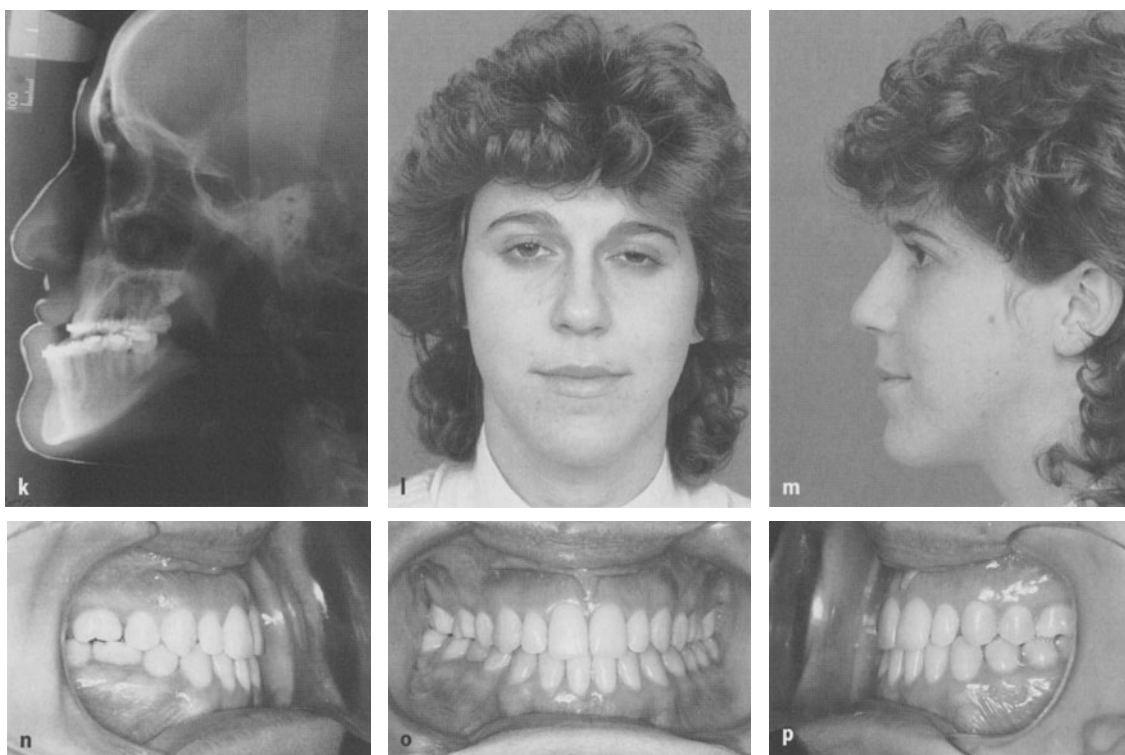


Fig. 61k-p. Bilateral H.E., slender/non-slender type with circular open bite in a case of medium degree of micro-retromaxillism. **k** The lateral cephalogram after presurgical orthodontics now shows the actual relationship between the upper and lower teeth in accordance with the length of the jaw bases. The patient's **l** front and **m** profile views 1 year after correction by a mandibular setback and a maxillary advancement operation, showing a good profile line and facial contour. **n-p** The patient's occlusion 1 year after surgery

romaxillism. And just as important is: b.) “Do not extract teeth when there is enough length of the base of the jaw to place all of them into correct angulation to it”. In this case that is true for the mandible. It will increase the discrepancy between the upper and lower dental arches. But it will better enable the surgeon to move the bases of the jaws to where they need to be in order to produce a good profile. c.) “Form two dental arches which will fit together”. That principle is valid for any presurgical orthodontics.

2. After the presurgical orthodontic preparation has achieved what is wanted, a bilateral sagittal splitting procedure on the rami with mandibular retropositioning and additional advancement of the maxilla should achieve the planned occlusion and correction of the facial anomaly.

The planning for the surgical correction was done as usual.

Actual Treatment. The presurgical orthodontic treatment (Dr. G. Ferrazzini, Lugano) produced quite an an-temandibulism appearance (Fig. 61k). The surgery was carried out according to the plan, by one of my trainees. The illustrations in Fig. 61l–p show the result 1 year after surgery.

Discussion. This case of H.E. demonstrated clearly that one should also always have tomograms of the TMJ–ra-

mus region for a proper diagnosis. Without these tomograms, my diagnosis, based on the panoramic view only, would have been a bilateral H.E. of almost a non-slender type. Although that diagnosis would not have made any difference in the treatment plan, nevertheless it would have been incorrect.

The case also demonstrated that in a case of bilateral H.E. a circular open bite may develop. The open bite develops due to a downward bowing of the body in the bicuspid region on both sides when the hyperactivity is not too asymmetrical.

In addition, this case proves the necessity to have, and follow, clear principles for the presurgical orthodontics.

Conclusion. There are different types of unilateral H.E., as there may also be some individual bilateral cases. Any gradation from the pure slender type to the pure non-slender type can exist. For the treatment plan, this differentiation does not make much difference except for the angle area and the height of the body. The pure slender types have very obtuse angles or almost no angles at all. The pure non-slender type will have an almost normal condyle and neck and also a rather good angle and body height in the molar region. In the bilateral type, a circular open bite may develop due to a downward bowing of the mandible in the premolar regions. The open bite will only be circular and nearly symmetrical when the bilateral L-hyperactivity is not too asymmetrical.

Bilateral H.E., Slender Type, Commencing After Normal Skeletal Growth has Ceased (Fig. 62)

Sex and Age. Male, 28 years of age.

Aetiology and History. The patient was already 20 years old when he realized that the chin was growing further forwards. He had no idea why this had started. The forward development continued until the age of 26 years. There was nothing similar known in the family history. He developed TMJ clicking and occasional pain. This was the reason for his referral for surgery on his protruding mandible.

Clinical Findings. The patient has a markedly protruding mandible. It also seems large in its circumference. The right angle area is more prominent than the left. The mid-facial area seems flat and he has a big nose (Fig. 62a,b). The prominence of the chin is a little out of the facial midline to the left. Musculature and facial nerves are functioning normally. Mouth opening is normal.

On closing the teeth there is a massive negative overjet of 10 mm (Fig. 62c–e). Only the last molars occlude. The lower midline is shifted over to the left by about 3 mm.

Radiographs. The panoramic view of the jaws (Fig. 62f) shows that the mandible has clearly elongated condylar necks on both sides. The right is slimmer than the left and perhaps also a bit longer. Both angles seem rather stretched although there is some bony mass. The deviation of the midline of the lower incisors is also very clearly to the left, but probably partially corrected by the slight mouth opening.

The TMJ tomograms showed the condyles to be normal in shape and size, the right one a bit more voluminous.

The p.a. projection of the mandible in the open mouth position showed nothing remarkable apart from a slightly more prominent right angle region and the elongated condylar necks.

The lateral cephalogram (Fig. 62g) shows clearly the very long mandible with the two rather stretched angles and a good-sized maxilla in normal position.

The measurements of the tracing of the panoramic radiograph (Fig. 62h) do not disclose any large differences

between the right and left sides except in the articular processes. The left is 5 mm shorter, its neck 2 mm narrower and its condylar width is 2 mm greater than the right. Both angles are slightly larger than normal; the right measures 133° and the left 135° . The length of the horizontal and ascending rami is almost the same on both sides.

Provisional Diagnosis. Bilateral H.E., medium-slender type, which had started to develop after normal skeletal growth had ceased.

Treatment Plan. As the model operation showed that a very satisfactory occlusal result could be achieved with a mandibular set back operation, no presurgical ortho-

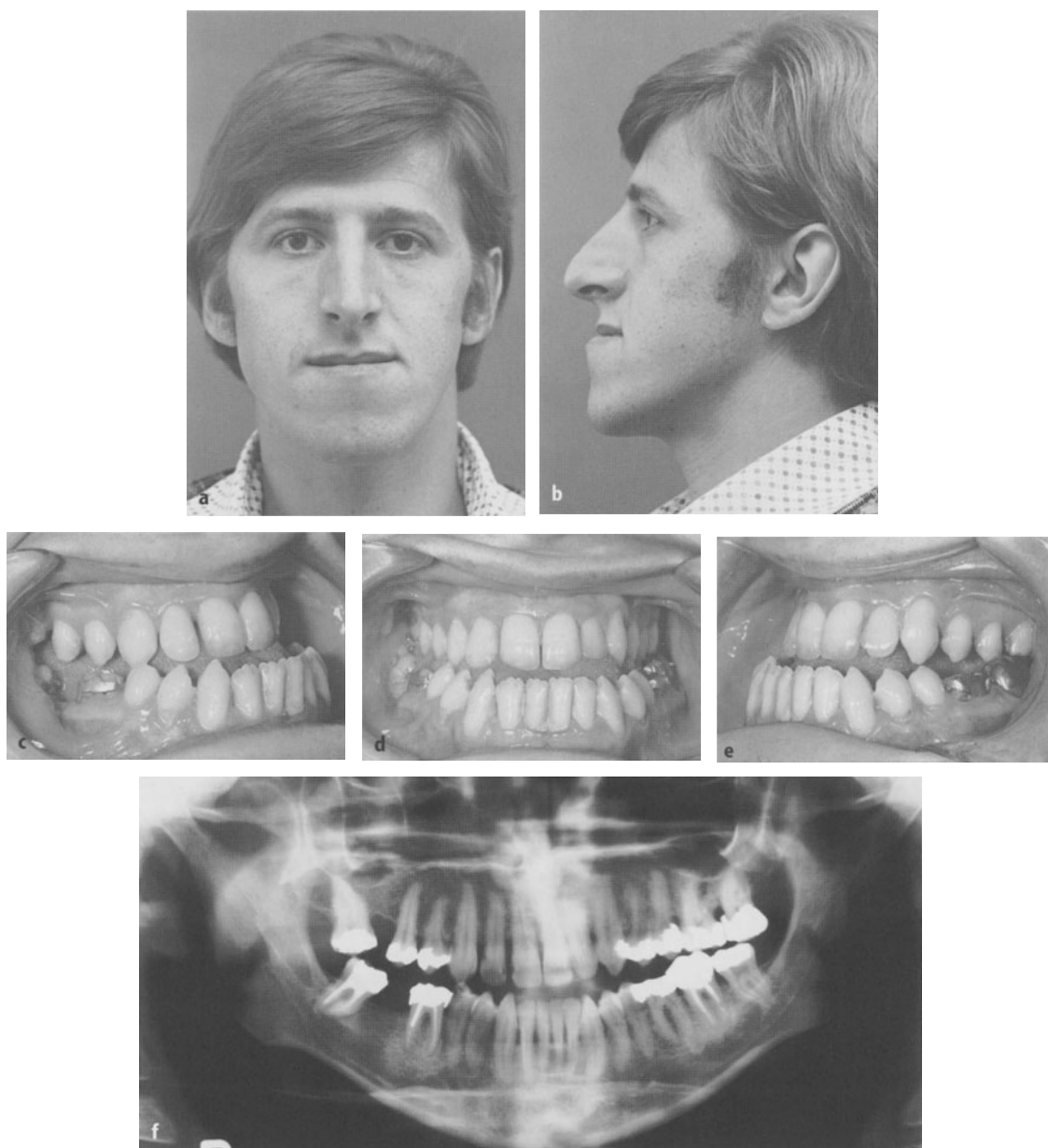


Fig. 62a–f. Bilateral H.E., developing after normal skeletal growth has ceased. **a, b** Severe antemandibulism with a large mandible, slightly asymmetrical, and some flatness of the middle third of the face. **c–e** The occlusion is similar to any severe antemandibulism case. Very slight deviation of the lower teeth midline only. **f** In the panoramic view the long condylar necks are striking, otherwise the strong mandible seems almost symmetrical and unremarkable

dontics seemed indicated. The usual profile planning did not indicate any necessity for repositioning of the maxilla. A bilateral rami sagittal splitting operation only was needed to achieve the result desired.

Actual Treatment and Result. The surgery was carried out as planned with the lateral cortical cut directed towards the angles in order to reduce the wide flat angles and give a better shape. Circummandibular wires and 6 weeks intermaxillary fixation maintained the planned position until bony union had occurred.

The result confirmed the correctness of the planning. The lateral cephalogram taken 1½ years after the corrective surgery (Fig. 62j) shows a normal skeletal relationship for a vertical profile type. The angles of the mandible have improved by 14°. This skeletal background is also seen in the patient's front and profile

views (Fig. 62j, k) taken over a year after surgery. There is a normal occlusion without any necessity for pre- or postoperative orthodontics (Fig. 62l–n).

Discussion. Looking at this case clinically, without knowing that the mandible grew forwards only between the ages of 20 and 26 years, one could easily have assumed that it might have been an ordinary inherited or anlage-induced antemandibulism case. The shape of the two hemimandibles was very similar and there was almost no asymmetry. The small deviation of the incisor midline could also have been due to poor tooth position. However, the fact that forward growth of the mandible started after general growth had ceased did not support the cause being a genetically determined or an inherited anomaly. These would grow gradually forward during normal skeletal growth. Only the additional

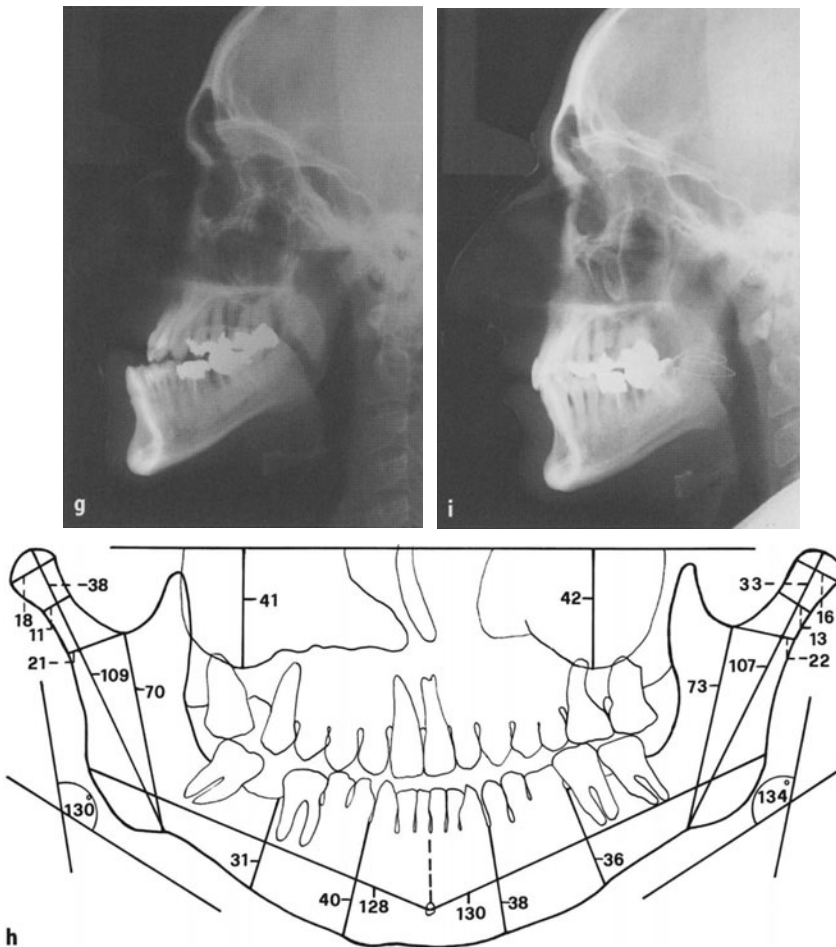


Fig. 62g–i. Bilateral H.E., developing after normal skeletal growth has ceased. **g** The lateral cephalogram shows that this massive very protruding mandible has rather stretched angles. **h** The measurements of the tracing of the panoramic radiograph confirm what it appears to show. **i** The lateral cephalogram taken 18 months after surgery shows a good facial skeletal relationship for a vertical profile type. The angles have improved remarkably

stimulation of the condylar growth factors can make the mandible grow after normal growth has ceased. This is true for the unilateral as well as the bilateral cases. Also, the difference in the condylar necks indicated that different growth activity was the cause of it. Although in this case there was only a little shift of the midline to one side, it automatically raises the suspicion of its being of H.E. origin. It could only be the case that both sides were influenced almost equally by hyperactivity of the L-regulator only. The difference in size and shape of the condyles and their necks automatically suggested that the local steering mechanism was the cause of this slightly asymmetric antemandibulism.

There was another interesting fact to be noted. In spite of the clearly H.E. slender type condylar necks with stretching of the angles, there was still a rather prominent bony angle on both sides and also fairly good mandibular body height in the molar regions. This was clearly contrary to the shape of the angle and height of the body in cases in which the H.E. slender type developed during puberty or even earlier. So it might be assumed that the patient had an absolutely normal shaped mandible until the bilateral L-hyperactivity

started at the age of 20 years. This could then still have elongated the ascending rami and stretched the angles, but could not have eliminated the already existing bony mass of the angles.

The other possible cause of a mandible becoming so large and elongated after growth has ceased could only be acromegaly. But neither the patient's face nor the radiographs showed any signs of the anomaly being acromegaly.

Conclusion. The differential diagnosis between a true genetically predetermined antemandibulism (prognathism) and a bilateral almost equal H.E. type of antemandibulism may be very difficult. It seems easy when the overgrowth of the mandible starts after normal growth has ceased and when there is a clear difference in size and shape of the two condyles and their necks. It also seems that a late-starting, H.E. slender type anomaly may not produce the classic slim angle regions as when it develops in childhood. The already developed mass of bone in the angle region remains in spite of the stretching of the angle.

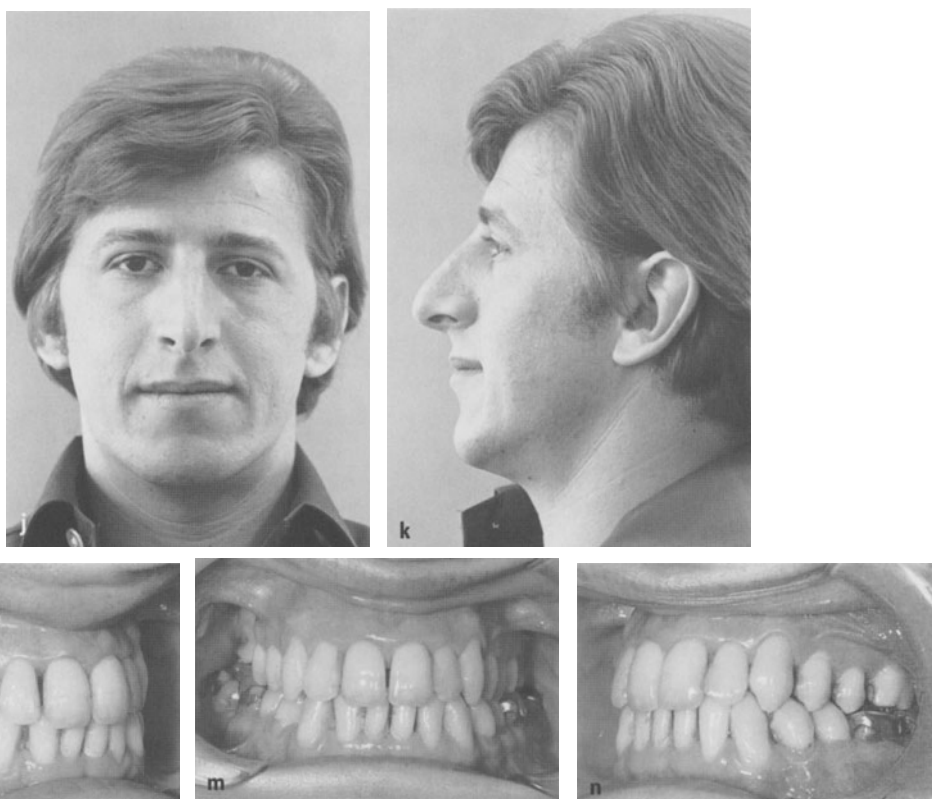


Fig. 62j–n. Bilateral H.E., developing after normal skeletal growth has ceased. **j, k** The patient's front and profile views, taken 14 months after the mandibular setback operation by a bilateral sagittal splitting procedure on the rami. **l–n** The patient's final occlusion, also 14 months after surgery

Bilateral H.E., Non-Slender Type, Producing a Severe Open Bite (Fig. 63)

Sex and Age. Male, 17 years and 7 months.

Aetiology and History. The patient related that his mandibular anomaly had already started when he was a small boy and had become gradually worse. He had no idea of a possible cause. There was nothing similar in the family history. He had suffered psychiatrically since childhood because of his facial and occlusal deformity. There had been no change for the past year.

Clinical Findings. The patient has a mandibular long face, with some flatness in the infraorbital-paranasal areas and a humped nose (Fig. 63a, b). The two hemimandibles seem very long and straight and produce a rather pointed chin, which is shifted over to the left by 5 mm. The lip commissure is horizontal. Mouth opening is normal.

Occlusion exists on the right side only between the second lower molar and the upper second bicuspid and on the left side between the lower second and the upper first molar (Fig. 63c–e). There is an open bite of 12 mm incisor distance, with the lower teeth quite a bit in front of the uppers. All teeth in the mandible are severely lingually tilted and there is severe crowding in the maxilla. There is an upward convex curvature of the lower occlusal surfaces (reversed curve of Spee). The midline of the lower teeth is over to the left by nearly the width of one incisor. The tongue is rather large. Looking at the patient from the side there is no antemandibulism (class III) profile.

Radiographs. In the panoramic view (Fig. 63f) the ascending rami seems normal in shape, width and length with almost normal condyles and necks. They produce a normal angle with the body on each side. In front of the angle the horizontal rami have a downward bend producing a very severe open bite. The body of the mandible is of normal height, also in the molar regions. The lower teeth midline is shifted to the left by nearly the width of a lower incisor. Only the muscular processes are particularly conspicuous. They are unusually small, almost rudimentary only, and nearly equal in size and shape.

The measurements of the tracing (Fig. 63g) of the panoramic radiograph do not show any clear differences between left and right. Both ascending rami are of the same length. This is not true for the two horizontal rami, the left being 7 mm longer than the right. Both angles are rather large, 136° on the right and 131° on the left side. So, the cause for the shift of the Pg-point over to the left side, in spite of the left horizontal ramus being 7 mm longer, can only be the larger right angle. The height of the horizontal rami in the molar region is also almost the same.

The TMJ–ramus tomograms (Fig. 63h, i) show a left condyle and neck of normal size, shape and length. The right condyle is a bit smaller and has a modestly elon-

gated neck with a broad base. The trabecular structure is normal.

The mandible in the p.a. projection with the mouth open (Fig. 63j) shows rather long ascending rami and severe lingual tilting of the lower teeth and crowding of the uppers, but also not much difference between the two sides.

The lateral cephalometric view showed a clear stretching of both angles of the mandible producing an almost straight line from the condyles to the chin and nevertheless with rounded angle prominences. The base of the maxilla was very short and in a dorsal position. The open bite, the hump of the nose and the strange angulation of the occlusal plane of the mandible cannot be overlooked.

The lateral cephalogram after presurgical orthodontics discloses even better the differences between the mandible and the maxilla and that there is not only a hypergenia but also a clearly elongated middle third of the face (Fig. 63k). The evaluation of its tracing proves again that the panoramic radiograph is not suitable for measuring the real values of the angles. On the tracing of that radiograph the angles measure 136° on the right side and 131° on the left, while the lateral cephalogram produces the correct figure of 143°.

Provisional Diagnosis. Bilateral H.E., non-slender type, producing a severe mandibular open bite deformity. In addition there was a micro-retromaxillism with severe crowding of the upper teeth.

Treatment Plan. The planning of the treatment was done in the way we usually did it, at least for the severe anomaly cases: the patient's profile photograph is enlarged to actual size by the use of a tracing of the profile line of the lateral cephalogram. On the large photograph a piece of transparent tracing paper is fixed. On this the desired profile is drawn, usually by an artist, and transferred to the photograph. The case is then discussed with the orthodontist with the new profile plan. At the Zürich Dental School I had the privilege of discussing such cases with Paul Stöckli, the chief of orthodontics. We often discussed problem cases and treatment principles. I am glad he agreed with the principle of presurgical orthodontics which I formulated as follows: "The aims of presurgical orthodontics must be:

- a) To arrange the teeth in proper angulation to the jaw base, independent of the number of extractions necessary
- b) Not to extract teeth when there is enough length of the base of the jaw to place all of them into correct angulation to it
- c) To create a normal curve of Spee

d) *To create dental arches which will fit together*" (Principle No. 25)

As the patient was from far away, he sent his orthodontic treatment plan to a former pupil of his, Dr. D. Grobety in Vevey, who kindly did the necessary presurgical orthodontics very efficiently.

When the patient returned for surgery 2 years later there was now a severe antemandibulism (class III) ap-

pearance and a severe negative overjet (Fig. 63k), clearly demonstrated by the lateral cephalogram. For profile planning, the patient's new profile photograph was enlarged to natural size with the help of the tracing of the cephalogram. For planning the correction, the desired profile was drawn on a sheet of tracing paper on top of his new profile line (Fig. 63l). Then the maxilla, the mandible and chin were cut out of another full tracing in order to be able to move them on the tracing into the de-

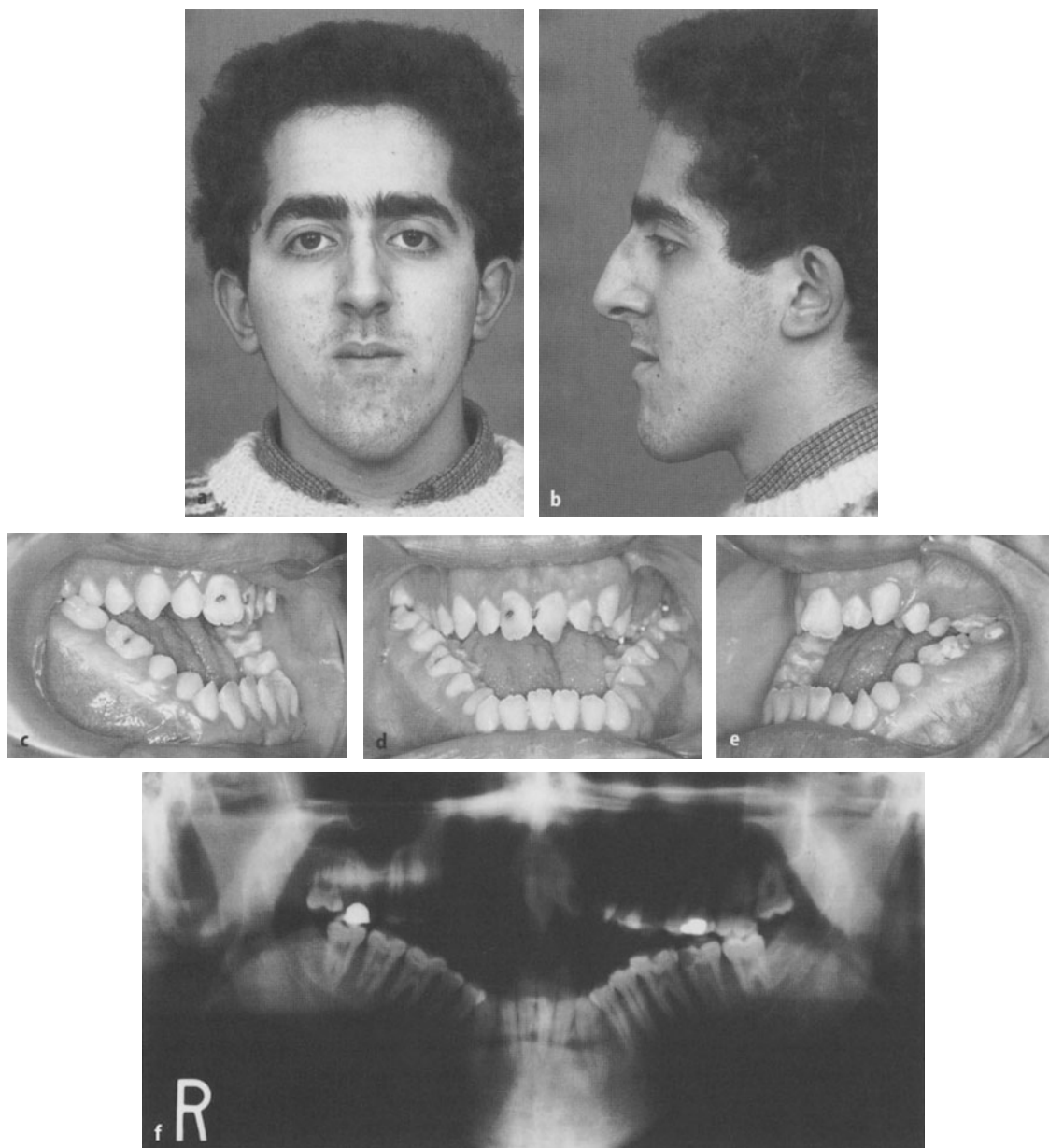


Fig. 63a–f. Bilateral non-slender H.E. producing a severe open bite. The patient's appearance in **a** front and **b** profile views. **c–e** The patient's occlusion with a severe open bite. **f** The panoramic view discloses that the open bite is mainly due to a kink in the pre-angle are-

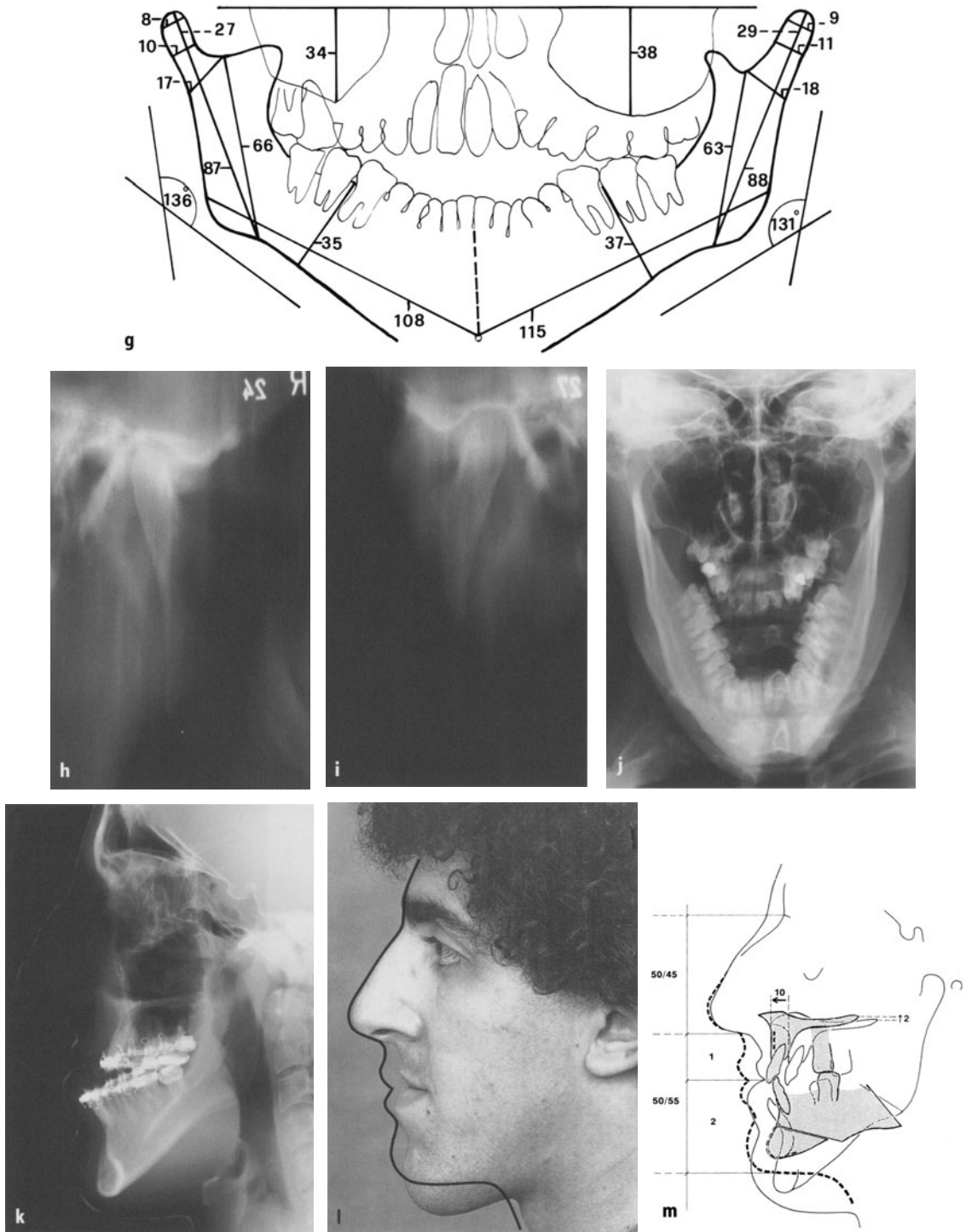


Fig. 63g-m. Bilateral non-slender H.E. producing a severe open bite. **g** The tracing of this radiograph cannot give the answer to the question why that kink has occurred. **h, i** The TMJ-ramus tomograms do not disclose anything of note. **j** The mandible in p.a. projection does not disclose any great difference between left and right sides. Both ascending rami seem remarkably longer than normal. **k** The lateral cephalogram after presurgical orthodontics demonstrates the enormous contrast between the long and straight mandible and the repositioned maxilla with its short base. **l** Original and planned profile lines. **m** The planned profile superimposed on the cephalometric tracing. Maxilla, mandible and chin moved into the desired position

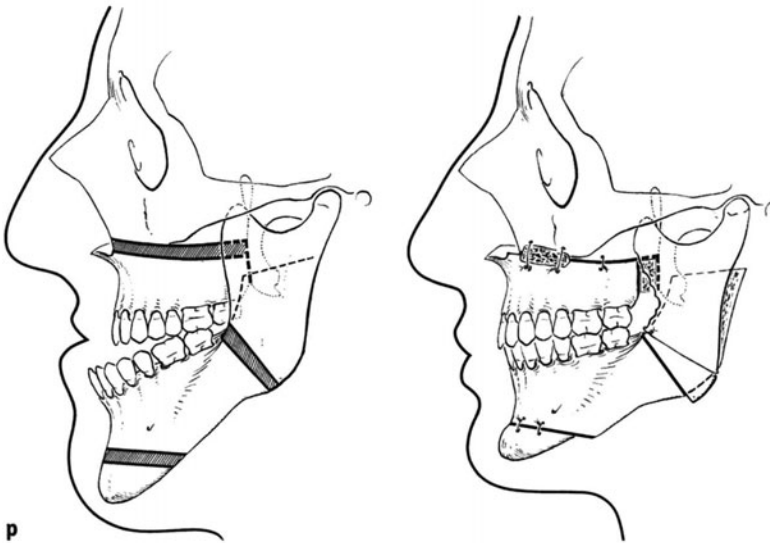
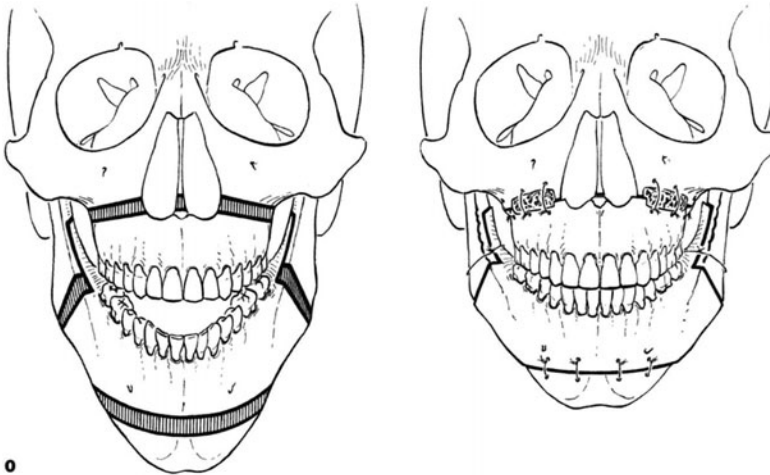
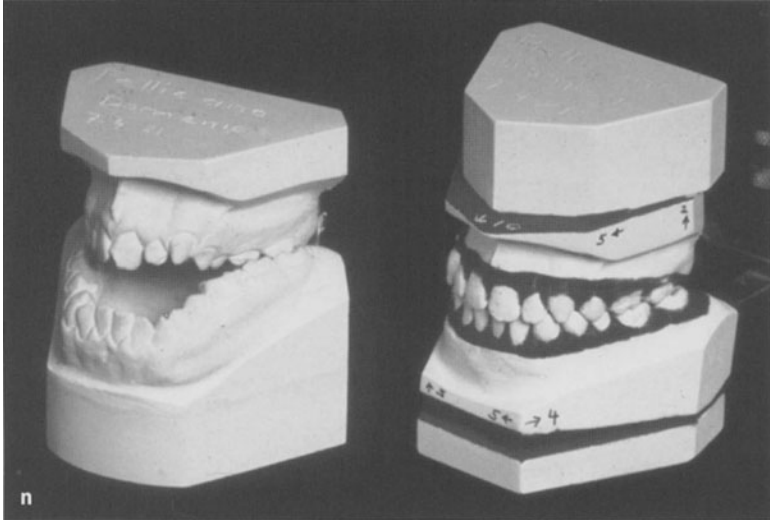


Fig. 63n-p

sired final position, which should produce the planned profile (Fig. 63m). That permitted transfer of the measurements of movements of the jaws to the model operation (Fig. 63n).

The maxilla needed to be raised by 5 mm and advanced by 13 mm. Next the mandible should be repositioned into correct occlusion using a bilateral sagittal splitting procedure on the rami. Furthermore the chin prominence should be reduced in height by a slice osteotomy of 7 mm and advanced, as the patient had requested a rather strong chin (Fig. 63o, p).

Actual Treatment and Result. The surgery was carried out in accordance with the plan as a routine procedure, directing the lateral cortical cut in the sagittal splitting procedure of the rami directly to the angles of the mandible in order to create proper angles out of the very stretched ones.

The postsurgical panoramic view of the mandible taken 13 months after the surgical correction revealed a mandible of normal configuration and symmetry. The preoperatively measured angles of 136° and 131° had changed on both sides to 122° .

The lateral cephalogram, taken at the same time (Fig. 63q) now reveals a normal facial skeleton producing a vertical profile type. The change in the angle measured 129° instead of 143° presurgically. That proves once more that the figures of the angles measured on the panoramic radiographs differ quite a bit from the measurements on the lateral cephalograms.

Due to the very efficient presurgical orthodontics it was a pleasure to produce a very satisfactory result in the patient's appearance as well as in his occlusion (Fig. 63r–v).

Discussion. Diagnosing such a case as a bilateral H.E. seemed risky, as the condylar growth regulator L produces lengthening of the affected side. But this case had bilateral lengthening and also a very severe open bite deformity. In unilateral H.E. we have encountered an open bite situation not only on the side of the lengthening, but also on the contralateral side, obviously depending on the growth force of the hyperactivity. In bilateral H.E. cases an open bite situation seems almost common. But could this very severe open bite case with an inferior bowing of both mandibular bodies have been produced by a bilateral H.E.? Any asymmetry of

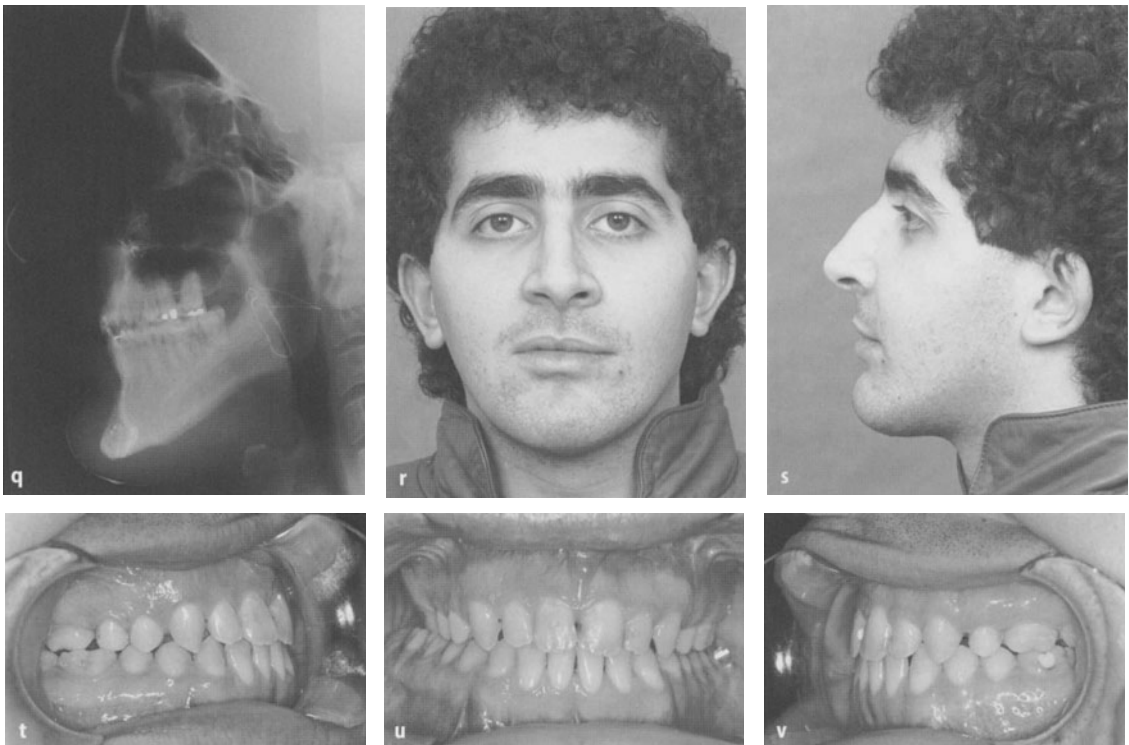


Fig. 63n–v. Bilateral non-slender H.E. producing a severe open bite. **n** Model operation including presurgical orthodontics. Diagrammatic illustration of planned surgical correction, in **o** front and **p** lateral views. **q** The lateral cephalogram 13 months after surgery reflects the new facial skeleton and permits its comparison with the preoperative facial skeletal situation. **r, s** The patient's appearance 13 months after surgery. **t–v** The final occlusion, also 13 months after surgery

the midline of the lower teeth and the chin is suspected of being the result of condylar growth misregulation, either hypo- or hyperactivity. If this case of bilateral elongation with its severe open bite was not the result of that; what else might have been the aetiology? I have seen such open bite cases due to neuromuscular problems or severe scarring of the neck and various other origins. They all relapsed after surgical correction when the cause could not be eliminated beforehand. If such an open bite develops due to muscular imbalance, relapse is to be expected.

Did the peculiarly small size of the coronoid processes have something to do with the aetiology of this very severe open bite? There comes first the question why are these muscular processes so small? On palpation, there was nothing special to be found in the functioning of the two temporalis muscles. If it were of muscular origin then it would still be active after the surgical correction of the open bite and consequently lead to a relapse within a few months. But nothing happened. So there is still no other explanation for that bilateral asymmetric elongation of the two hemimandibles with that severe open bite, but a bilateral H.E.

I have experienced complete relapse of a set back of the mandible, in which the still persisting activity of acromegaly was not diagnosed presurgically although the patient had been sent for endocrinological investigation of the possibility of such an abnormality. The bad experience with all such cases can be summarized in two

principles: *"If you do not remove the persisting cause of a facial skeletal anomaly, the relapse of your corrective surgery is preprogrammed"* (Principle No. 31) and *"The active cause of a facial skeletal anomaly is stronger than screws and plates. Plates and screws cannot prevent a relapse if the cause of the anomaly is still active"* (Principle No. 32). These principles are true also for cases of condylar hyperactivity and probably for hypoactivity cases too. But when the hyperactivity is burned out there should be no risk in the surgical correction. In this case the hyperactivity had already subsided a year before the patient asked for correction and 3 years before surgery, as the presurgical orthodontics took almost 2 years.

My thesis is no real proof of this case being the result of a former bilateral condylar hyperactivity. The only real proof could be a higher than normal RU-value on scintigraphy during the active phase. Another supportive proof could be the histology from a specimen obtained preferably also during the active phase. Both possibilities are gone once the abnormal growth has ceased. There seems to be no other way to come to an acceptable aetiological explanation in such cases than via a diagnosis by exclusion.

Conclusion. Bilateral H.E. can even produce a very severe open bite mandibular deformity. As long as the cessation of the hyperactivity is confirmed and proper treatment planning and execution is carried out, then there should be no risk of relapse.

13.3.1 Differential Diagnosis of Bilateral H.E.

First of all, an anlage-induced antemandibulism might be diagnosed as a bilateral H.E. But that real antemandibulism is symmetrically equal in length and shape of both sides of the mandible. This is rarely or never true for a bilateral H.E.

In the case of retromaxillism with a slight rotation, or where there is a pure dental cause for the upper teeth midline being out of the facial midline, such a finding might be misdiagnosed as a bilateral H.E. For that reason it is always necessary first to determine the facial midline. Normally it is recognizable in a p.a. cephalogram by the position of the crista galli, also of the nasal crest of the palate and, usually, also the position of the nasal septum.

Whenever I am in doubt about facial asymmetry, to ascertain the truth I will use the procedure advocated in the following principle: *"When you are not sure whether a facial asymmetry exists or not place the patient's head in an upright position, then, with a pencil, draw a horizontal line above the eyebrows and to that an imaginary line perpendicular to the middle of the forehead. It can then be seen clearly whether a facial asymmetry exists or not and what is on which side of the face and what the differences are between the two sides of the face"* (Principle No. 16). By adhering to this principle then one can

easily determine whether or not the teeth midline is congruent with the facial midline. The same is true for the chin prominence, the zygomatic bones and, particularly, for the nasal bridge.

Apart from the desired asymmetry in a case of bilateral H.E., there is another important fact which permits differentiation between true antemandibulism and bilateral H.E. The development of a true antemandibulism normally becomes noticeable in early childhood. It almost always becomes visible before the age of 6, while I have never observed or heard of the development of a true H.E., uni- or bilateral before the age of 5.

One could argue with the fact that there are cases of asymmetric antemandibulism, known to all of us, which obviously do have a genetic background. Should they all be diagnosed as bilateral H.E. forms in spite of the fact that in unilateral cases of H.E. no pertinent family history is known? Should unilateral H.E. not have a genetic basis while the bilateral H.E. could? Definitely not! What then is the explanation for the fact that bilateral asymmetric forms of antemandibulism with a clear family history are known? One explanation might be that in any case of bilateral symmetrical antemandibulism, an additional unilateral H.E. condylar hyperactivity can be superimposed.

Real Antemandibulism (Mandibular Prognathism) (Fig. 64)

Sex and Age. Female, 17 years and 2 months.

Aetiology and History. Nothing is mentioned in the patient's records about aetiology or a positive family history. The parents had already noticed the forward growth of the mandible early in childhood.

Clinical Findings. A good looking young lady with a completely symmetrical face (Fig. 64a). The lower lip is a bit in front of the upper. In the resting position of the mandible her lower third is too high (long). The lip commissure is absolutely horizontal and symmetrical. The lower border of the mandible is straight and a bit long (Fig. 64b). The angles are inconspicuous. The mouth opening is normal without deviation. The upper second bicuspid is missing, but the gaps are closed. The occlusal plane is horizontal. In occlusion there is a bilateral crossbite with a complete class III teeth relationship and a negative overjet of about 4 mm. The midline of the lower teeth is 2 mm to the left (Fig. 64c–e).

Radiographs. The panoramic view (Fig. 64f) shows a symmetrical mandible of normal shape in all its sections. The condyles are rather small, but rounded with a normal fine trabecular structure. The right neck seems somewhat elongated and the right angle seems to have a larger radius than the left, which is quite well shaped. All teeth are in an upright position without any tilting. The upper and lower midlines of the teeth are exactly in the facial midline. The trabecular structure of the mandible is fine, the horizontal and ascending rami are within normal limits and the mandibular canals run close to the apices of the teeth at a clear distance from the lower border. On both sides the body height is rather low. There is no downward growth of the maxilla on either side.

The measurements of the tracing of the panoramic view (Fig. 64g) prove that both sides are equal in all sections, except for 2 mm differences in the vertical height

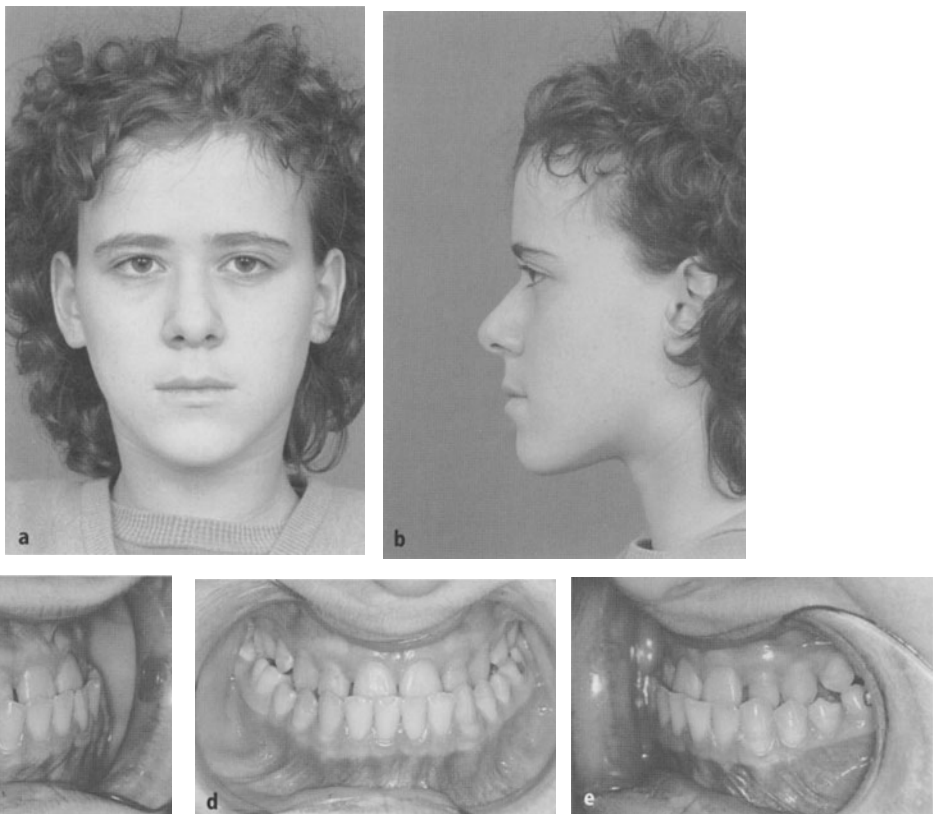


Fig. 64a–e. Real antemandibulism. The patient's **a** front and **b** profile photographs do not show any facial asymmetry. **c–e** The occlusion is typical of such an anomaly. The slight asymmetric lower teeth midline is not sufficient to diagnose a H.E. growth anomaly

of the horizontal rami in the molar areas. Both horizontal rami measure 107 mm, both ascending rami 75 mm, both trunks 53 mm and both articular processes 22 mm.

The TMJ–ramus tomograms (Fig. 64h, i) show nothing of note, just rather gracile condyles and necks and rather short rami. Everything is within the normal range, as is the right angle.

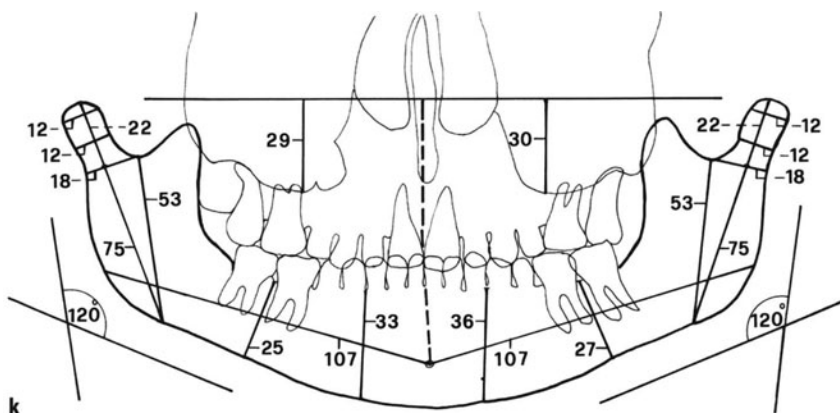
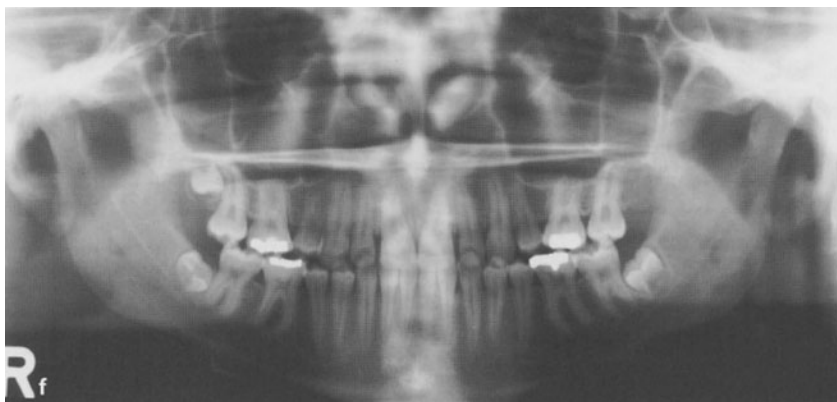


Fig. 64f–k. Real antemandibulism. **f** The panoramic radiograph does not disclose any asymmetry. **g** The measurements of the tracing of the panoramic view prove that both hemimandibles are equal in all sections. **h, i** The TMJ–ramus tomograms show that the condyles and their necks as well as the rami and angles are within normal limits in size and form. **k** The lateral cephalogram gives the true picture: the horizontal rami are too long compared with the other parts of the facial skeleton

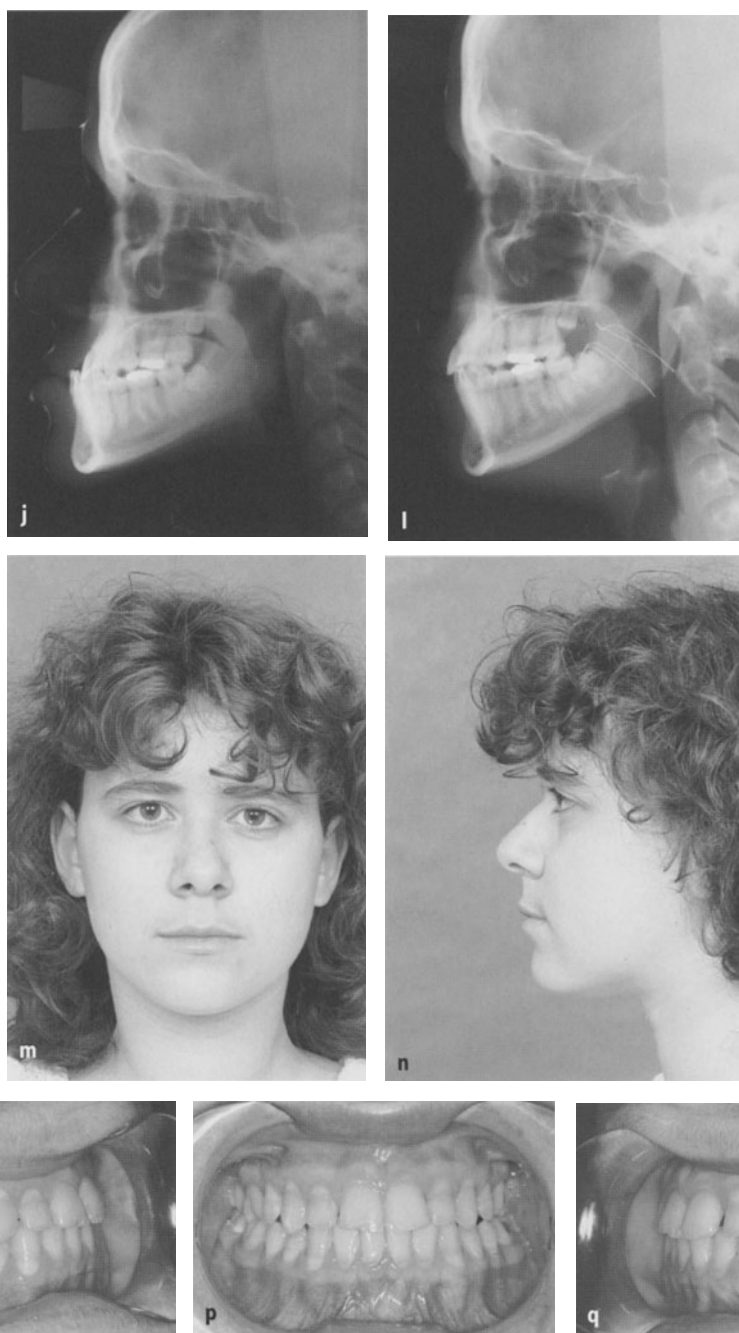


Fig. 64j, l–q. Real antemandibulism. **j** Also the mandibular p.a. view does not disclose any abnormality, except rather long horizontal rami. **l** The lateral cephalogram, taken 1 year after surgery, shows an absolutely ideal facial skeletal situation. It proves, in comparison with the presurgical lateral cephalogram, that the term “antemandibulism” described the presurgical situation correctly. **m–q** The patient’s appearance and occlusion 1 year after the transformation of the antemandibulism into a normomandibulism by bilateral sagittal splitting of the rami and mandibular repositioning

The mandible in *p.a.* projection in the maximum opening position (Fig. 64j) discloses a mandible absolutely symmetrical in size and shape, compatible with a female of 17 years of age. There is no elongation of the ascending rami noticeable.

The lateral cephalogram (Fig. 64k) discloses the typical skeletal appearance of antemandibulism with the maxilla of normal size and in a normal position (normomaxillism). The bodies of the mandible are too long for that facial skeletal picture, producing the clinically-observed negative overjet.

On the wrist radiograph, all epiphyseal sutures were closed.

Provisional Diagnosis. True pure antemandibulism.

Treatment Plan. Some presurgical orthodontics, followed by mandibular repositioning by the use of the bilateral sagittal splitting procedure on the rami.

Actual Treatment. The patient did not want any presurgical orthodontics since the model operation showed that a good occlusal relationship could be achieved without it. The operation was performed by a trainee at my clinic, with perimandibular wires and 6 weeks intermaxillary fixation.

The lateral cephalogram, taken 1 year after surgery, shows an ideal interskeletal relationship (Fig. 64l). The operation, pushing the mandible into the ramus, created a normomandibulism. The aesthetic result was very

pleasing without any additional surgery and the occlusion was also very acceptable (Fig. 64m–q).

Discussion. Many bilateral H.E. cases do produce an antemandibulism situation similar to a true antemandibulism which is not due to bilateral hyperactivity of the L-growth regulator, but due to the patient's genetic pattern or is anlage-induced. As long as there is no asymmetry and there are no other clear radiological signs of one of the H.E. types, we must call it true antemandibulism. It is as if the mandible had grown too far forward, from the angle areas. Therefore, it is logical that the result must be good if we push it back into them again by the use of bilateral sagittal splitting of the rami. In such a case it does not matter where the lateral cortical cut is placed, directed to the angle, or in front, or above it.

Conclusion. The differential diagnosis between bilateral H.E. of any of its types and true antemandibulism is the fact that the latter presents a normal shape and size in all parts of the mandible with the exception of the symmetrically too long bodies. As long as there is no skeletal asymmetry and no other signs of a H.E. type anomaly, I just call it pure antemandibulism in spite of knowing that there may be the very rare case of a bilateral H.E. which produces equally shaped hemimandibles and no other signs of a growth misregulation anomaly. If it were the result of symmetrical bilateral hyperactivity of the condylar L-regulator we would have to find signs of H.E. In this case there is no sign of it at all.

Hybrid (Mixed) Forms of H.H. and H.E.

The simultaneous hyperactivity of both the M- and L-condylar growth regulators in one condyle must produce a mixture of mass and length production on the affected side of the mandible. These hybrid cases can produce the most grotesque facial asymmetry disfigurement. Naturally, they may exist as very mild forms only or even as exorbitant facial malformations. Between these two extremes, any degree of hyperactivity of the two growth regulators may occur. One factor may be grossly predominant over the other. In this event either the H.E. type or the H.H. type is the cause of the leading feature of the clinical picture.

Any hybrid form must have clear features of both misregulation anomalies: there must be a crossbite, chin deviation over to the opposite side, and a vertical increase in the affected hemimandible with an oblique occlusal plane, easily diagnosable with a spatula between the teeth in occlusion, and there must be inferior bowing of the lower border of the mandible on the affected side.

The hybrid form should be diagnosed as early as possible. In the fast-growing case a high condylectomy must be performed without delay.

Unilateral Severe Hybrid Form Active for 13 Years (Fig 65)

Sex and Age. Female, 41 years of age.

Aetiology and History. The asymmetry had started at the age of 10 years and finished at the age of about 23 years. The patient had no idea why it had occurred. There was nothing similar known in the family history.

Clinical Findings. There is a very severe rotation and enlargement of the patient's mandible to the left side (Fig. 65a). The right side of the face is much longer than the left. The lower border of the right mandible is curved downwards and inclined medially. The chin is high (deep) and over to the left side by about 20 mm. The lip commissure is very oblique, ascending to the left. The left corner of the lip is much more lateral than the right. The profile views of the two sides show the severe elongation of the right side of the face with the enormously protruding chin (Fig. 65b, c). Musculature and facial innervation were normal. Mouth opening was not reduced. On maximum opening the chin returns towards the midline, but not completely (Fig. 65d).

When closing the teeth a severe crossbite on the left becomes apparent (Fig. 65e–g). The lower incisor midline is shifted excessively to the left. The right lower canine occludes with the midline of the upper teeth, the right lower first molar with the upper canine. The maxillary occlusal plane is almost horizontal.

Radiographs. The panoramic film (Fig. 65h) was not able to include the whole mandible. Nevertheless there is a clear step in the symphysis area. The left side is normal in shape and structure although rather short in height in the molar-bicuspid region. The right side is curved downwards with additional increase in the body height in the bicuspid region. The right angle is very rounded, with a large radius. The right ramus with its condylar neck is clearly longer than the left one. The neck of the right condyle is rather thick and on top of it a tumour-like rounded enlargement can be identified. The right mandibular canal is clearly visible in the ascending ramus but cannot be seen in the body region. There is a coarse trabeculation in the upper part of the right ramus. The maxilla seems positioned further inferiorly on the elongated right side.

The TMJ-ramus radiographs were stored at another Institute of the University Hospital. Since they were over 10 years old regrettably they had already been destroyed when I asked for them.

The lateral cephalometric projection (Fig. 65i) shows a rather grotesque picture of a human facial skeleton. The severity of the facial deformity seems to have two main components. One is the tremendous unilateral enlargement of the mandible in the vertical dimension

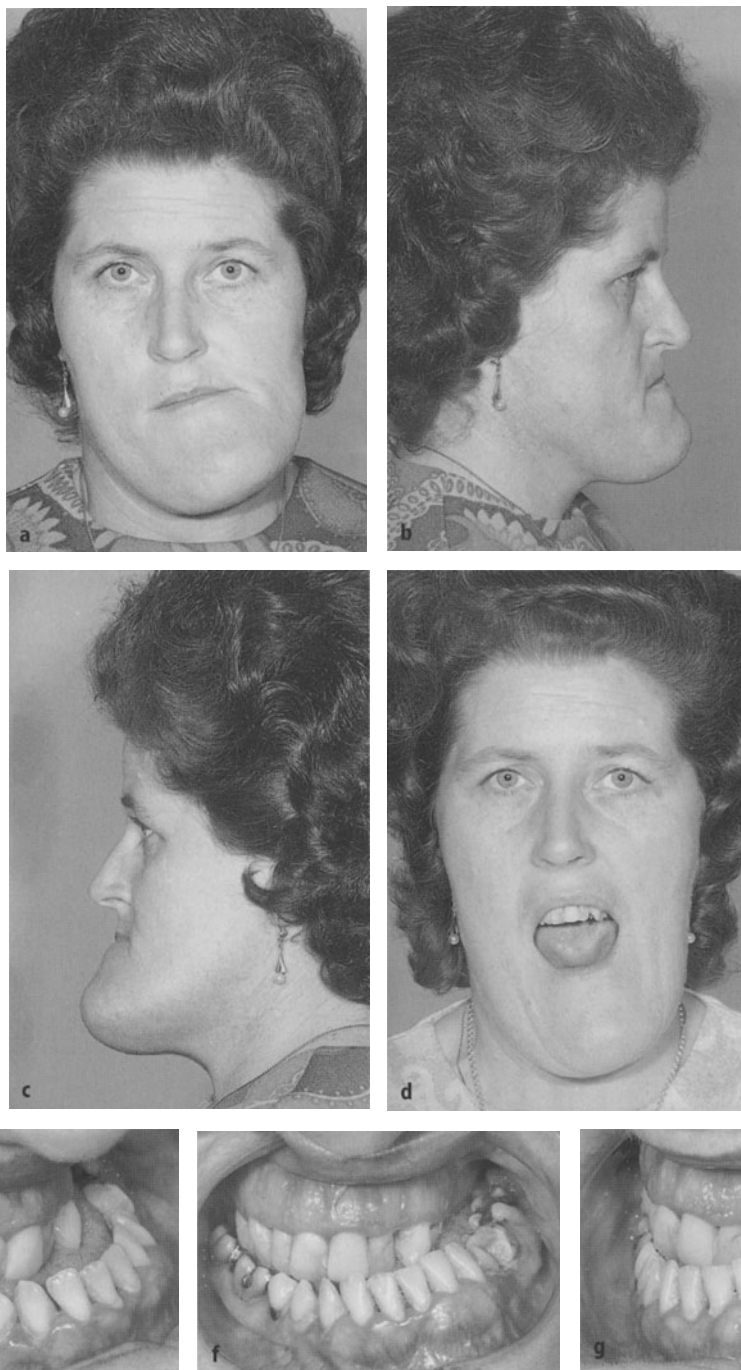


Fig. 65a-g. Unilateral severe hybrid form resulting from 13 years activity. **a-c** Typical front and profile appearance of a severe hybrid form of H.H. and H.E. **d** At maximum mouth opening the so severely asymmetric mandible comes almost back into the facial midline. **e-g** The classical occlusion picture, very typical of a hybrid form of H.H. and H.E.

and the other is the enormous elongation of the mandible in the sagittal direction. In contrast to this a severe skeletal dish-face deformity also seems to be present. One side of the mandible is much lower than the other side. The mandibular canal runs right down to the lower border in the bicuspid area. On top of the condylar neck sits a well-rounded tumour-like enlargement of the condylar head.

The *p.a. projection of the mandible* (Fig. 65j) shows the severity of the deformity of the mandible with a much longer right ascending ramus which deviates to the left side; there was lateral twisting of the left body. It is a unique picture confined to a very severe case of the hybrid form of H.H. and H.E.

Provisional Diagnosis. Mandibular asymmetry of uncertain origin (in 1968).

Today's Diagnosis. Unilateral excessive hybrid form of H.H. and H.E.

Treatment Plan. The patient was operated on by E. Steinhäuser when he was a senior co-worker of mine. He achieved a very nice result (Steinhäuser 1980, 1985).

Discussion. I had seen a very similar but even more severe case when I was a young trainee with my teacher Richard Trauner, in 1949. We did not then know what the cause was. When I saw this case in 1968 we did not

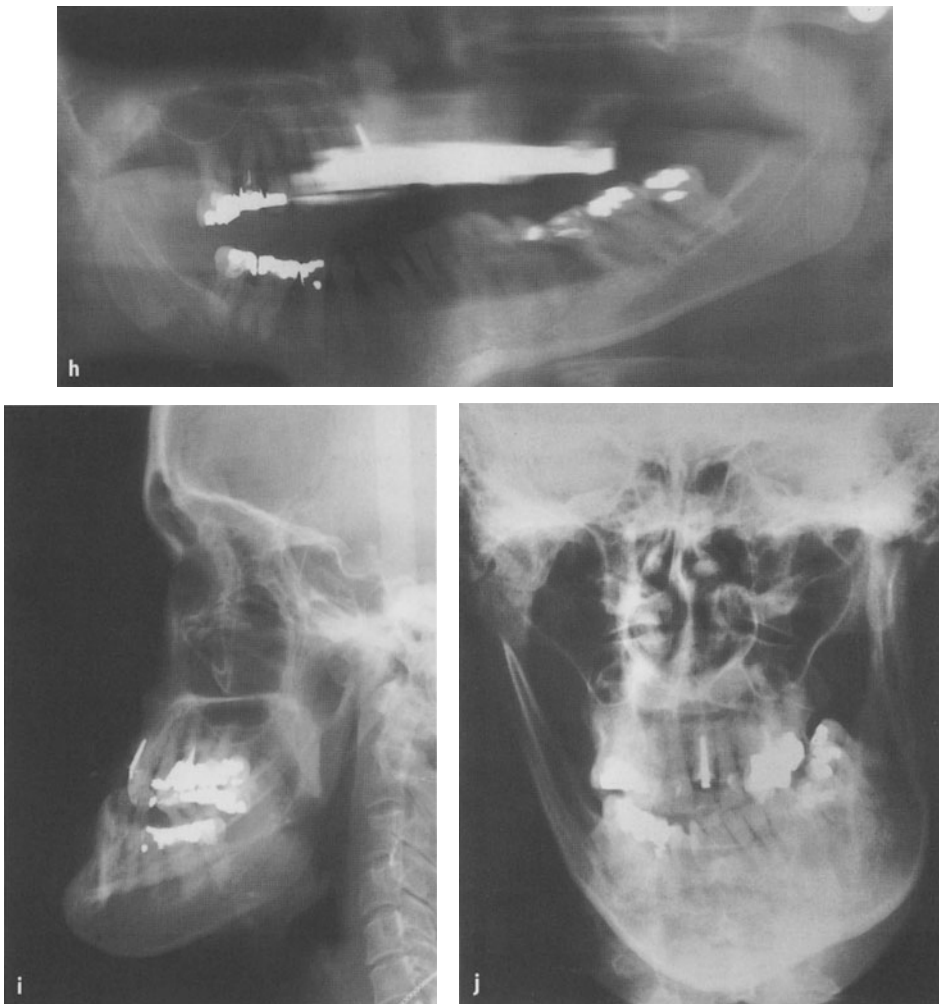


Fig. 65h–j. Unilateral severe hybrid form resulting from 13 years activity. **h** The panoramic view: the mandible is too large to fit onto the panoramic film. But what is on the film is sufficient to diagnose the growth abnormality. **i** The lateral cephalogram picture is very typical of the severe form of this type of growth anomaly. **j** The *p.a. projection of the mandible in the closed position* shows clearly the overproduction of length and mass of the right hemimandible with lateral rotation of the left horizontal ramus

know either! Only when I had seen enough pure H.H. and H.E. cases did I formulate the idea of the two different condylar growth regulators. As soon as that was clear and proven by early high condylectomy, then I could also understand this type of mandibular deformity resulting from simultaneous severe hyperactivity of the two condylar growth regulators in the same condyle. Although not every hybrid form may produce such a grotesque deformity of the mandible, this case does prove the necessity for early high condylectomy as soon as a patient starts to produce a rapidly developing hybrid form of unilateral H.H. and H.E.

Unilateral Questionable Hybrid Form with little H.E. Influence only (Fig. 66)

Sex and Age. Female, 22 years of age.

Aetiology and History. The patient had no idea why the mandibular asymmetry had started. There was no relevant family history either. At the age of 16 years she had noticed that the left side of her face became longer than the right and her chin had moved slightly out of the midline to the right side. She expressed the opinion that the asymmetry had not increased over the past 2 years. She had come for information regarding possible further behaviour of the asymmetry and what could be done.

Clinical Findings. Looking at the patient from in front (Fig. 66a), the facial asymmetry is very obvious with the left side remarkably longer than the right. The prominence of the chin is out of the midline to the right by about 6 mm, with its left side more prominent than the right. The right mandibular rim seems to be rotated outwards. Otherwise the cheek on that side has a normal contour. The longer left cheek is flat and its mandibular border is rotated towards the facial midline. The lip commissure is a little oblique with the left corner lower and more lateral than the right. Mouth opening is free with the chin prominence still remaining out of the midline to the right side (Fig. 66b).

There is a full dentition with an oblique occlusal plane tending to run downwards on the longer left side (Fig. 66c). Looking at the patient's face from below discloses even more the extensive difference between the two sides of the face (Fig. 66d). There are all the typical signs of a very well recognizable H.H. form: the difference in length of the two sides of the face is very impressive. The horizontal ramus of the longer side is tilted towards the midline of the face while that of the shorter side is rotated outwards. The chin seems out of the facial midline to the right. The two profile pictures (Fig. 66e,f) disclose the very remarkable difference in length of the two sides. In centric occlusion (Fig. 66g-i), the midline

Conclusion. The hybrid form of unilateral H.H. and H.E. can produce a more severe developmental mandibular deformity than any other aetiology, except tumours. The aim of the orthodontist, who is usually consulted first must be to find out by scintigraphy and clinical observation how much the abnormal activity is influencing that hemimandible. In any case of this type of deformity, occurring at any age, high condylectomy should be performed in order to avoid such a severe growth anomaly.

of the lower teeth is about 2 mm towards the shorter right side. There is a clear difference in the position of the lower premolars and molars between the left and right sides. Those on the longer left side are vertical whereas those on the right side are tilted lingually. The lower vestibulum on the right side is more laterally placed than normal and so are the lateral gingival margins of the lower molars and bicuspid.

Radiographs. The panoramic projection of the jaw in the closed position (Fig. 66j) shows clearly the length difference between the left abnormal and the right sides. The trabeculation structure is almost equal on both sides, but somewhat wider and slightly stronger in the bicuspid area on the longer side. The mandibular canal runs at a fairly good distance from the lower border. The cortical plate is clearly thinner on the longer left side, less than normal. The right body height is smaller than normal, in particular in the molar region. The angle on the shorter side has a denser bone structure than the one on the elongated side. Both angles are smaller than normal in their curvature. The lower border of the horizontal ramus of the longer side is clearly a bit convex and has no antegonial concavity, while the lower border on the opposite side has a normal pregonial flat concavity and is almost in a straight line. The symphysis is shifted to the shorter side by 3–4 mm. The lower front teeth are tilted towards the longer side, bringing the midline of the lower teeth almost exactly opposite the midline of the upper teeth, thereby compensating for the shift of the mandible to the shorter right side.

The difference in size and shape of the two condyles is very striking. The right one is absolutely normal while the left seems enlarged in all its dimensions. The enlargement includes the neck almost as far down as its base. Also the structure of the condyles differs. The right one is unremarkable while the left has no cortical cover-

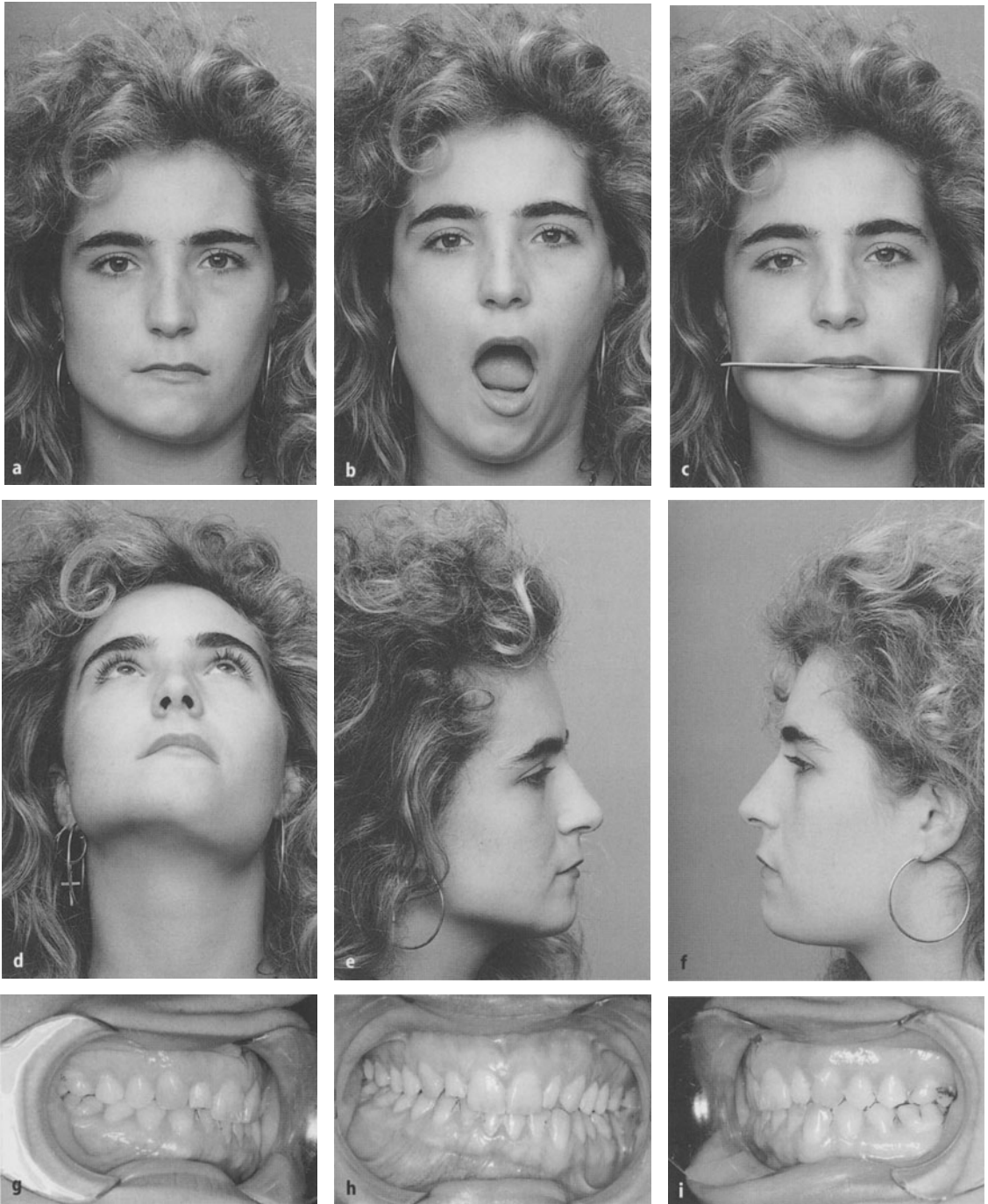


Fig. 66a-i. Unilateral hybrid form. **a-f** Appearance of a facial deformity due to a questionable unilateral hybrid form of condylar growth misregulation with only very little H.E. influence. **g-i** The occlusion is mainly that of a H.H. form, because of its marked predominance

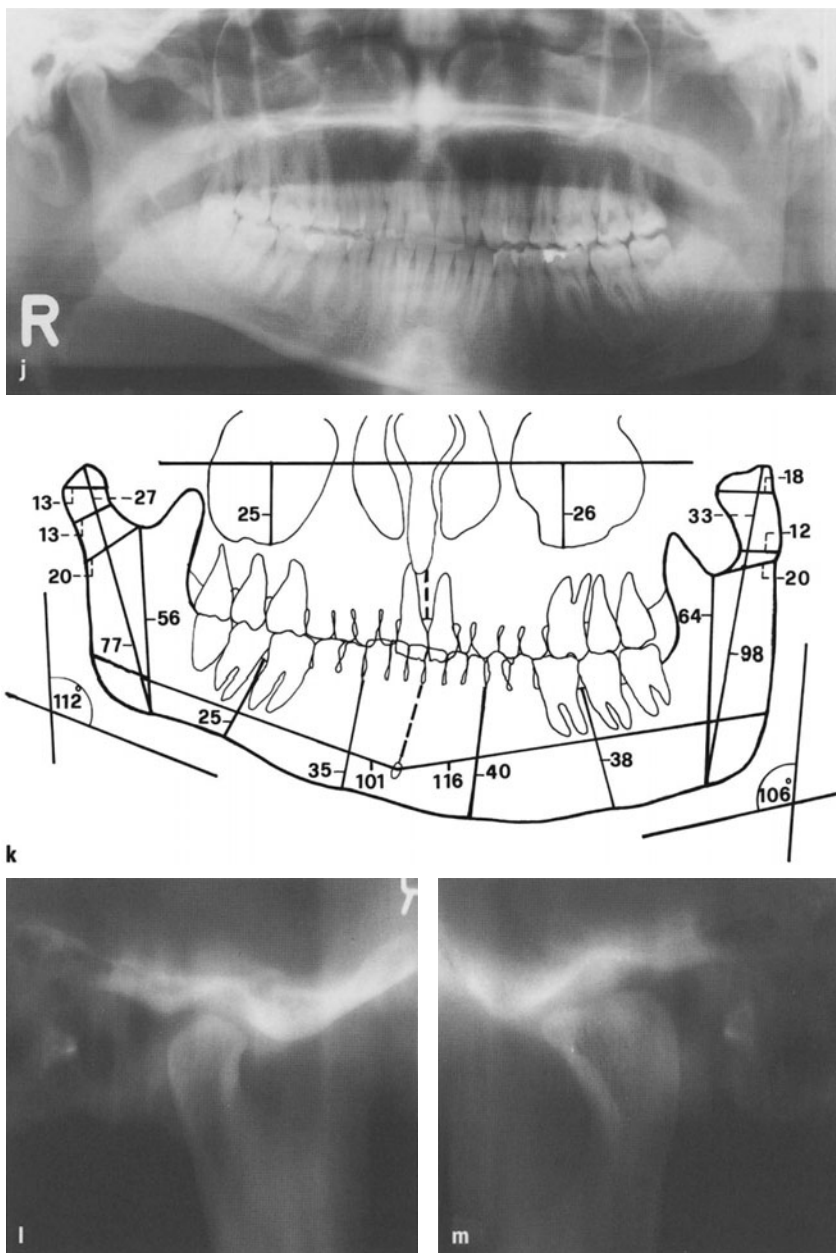


Fig. 66j-m. Unilateral hybrid form. **j** The panoramic view looks very much like that of an unilateral H.H., but the Pg-point and with it the symphysis is somewhat over to the normal side and the abnormal structure of the condyle extends rather far down into its neck. **k** The tracing of the panoramic view proves that all parts of the affected side are involved in the growth deformity and that the Pg-point is clearly out of the midline towards the shorter side. **l, m** The TMJ tomograms reveal a normal right condyle and fossa and a quite enlarged left condyle with an anterior “beak” and a coarse trabecular structure far down into its neck

ing, some parts are almost sclerosed and there is a rather large translucent area in the enlarged part of its neck. The shape of the enlarged left condyle is very typical of that found in H.H. cases but the elongation and thickening of its neck is an additional component, normally only found in H.E. cases.

The hybrid forms seem to include in their condyle enlargement the greater part of the upper neck, while the pure H.H. condyle deformity is limited mainly to the condyle itself.

The maxilla had grown downwards by 7 mm on the left side in the molar region.

The measurement of the tracing of the panoramic view (Fig. 66k) discloses very impressively that the left hemimandible has the typical shape of a H.H. type deformity but also that the Pg-point is over to the shorter right side by some millimetres.

The difference in length of the ascending rami, including condyles is 21 mm. The body height in the first molar region on the longer side is 28 mm compared to 14 mm on the other side.

The TMJ tomograms without the rami (Fig. 66l, m) show the typical enlargement and deformity of the left condyle of a H.H. case with its almost flat surface, the anterior "beak" formation and the flattened glenoid fossa, the latter very typical of H.H. cases. But it cannot be overlooked that the clearly coarse trabeculation of the condyle reaches inferiorly almost to the base of the neck. And it can also not have been overlooked that there is no border between the enlarged condyle and the thickened neck.

As the ramus is, regrettably, not included in these radiographs they do not show the full picture of the hybrid form. The panoramic radiograph shows it better.

The mandible in p.a. projection at maximum opening (Fig. 66n) shows clearly the tremendous length increase in the whole left side and the outward rotation of the "normal" side. It also discloses the rather coarse trabeculation of the enlarged left condyle.

The lateral cephalometric view (Fig. 66o) discloses the height surplus of the left side even better than the other projections. It again measures 20 mm in the first molar region. There is no abnormality visible in the patient's profile.

Scintigraphy was performed using 600 MBq Tc-99m-DPD. It revealed clear hyperactivity in the enlarged left condyle (Fig. 66p) compared with the inactive right side.

Provisional Diagnosis. Still active unilateral hybrid form of H.H. and H.E. with very pronounced predominance of the H.H. type.

Treatment Plan. The patient was offered high condylectomy on the left side and rotation of the lower half of the facial skeleton with repositioning of the maxilla, the left

side higher by about 6 mm and the right side lower by 2 mm. Furthermore, the height surplus in the left body had to be transferred to the right lower border after freeing the mandibular nerve.

Actual Treatment. None. The patient had come from another country and did not want to be operated on in a country foreign to her.

Discussion. This case was very interesting from several points of view. Primarily one might have diagnosed a H.H. straight away. However, the shift of the chin prominence to the opposite side did suggest an additional influence of the H.E. type although the midline of the lower teeth was almost congruent with that of the upper. But the panoramic projection disclosed that this was due to the tilting of the lower front teeth to the abnormal side. In a pure H.H. case the lower tooth midline always seems to be slightly towards the elongated side, because of the tilting of the teeth. In this case it is slightly over to the shorter side, in spite of the tilting. That suggests a certain H.E. influence in addition to the H.H.

There was no real crossbite, although the H.E. influence should produce it. But the lingual tilting of the cheek teeth easily compensated for a minor degree of crossbite. A look at the patient's face from below permits a clearer diagnosis of the position of the chin than when looking at the face from in front. But this can be misleading. To be certain one has to evaluate the facial symmetry properly by the previously described techniques: either directly on the patient's face or better on the natural size enlargement of the frontal photograph or even more precisely, as far as symmetry of the skeletal parts is concerned, by evaluating the tracing of the p.a. cephalogram (see Fig. 68n).

Another interesting point was the fact that there was still very clearly hyperactivity in the enlarged condyle although the patient was of the opinion that the abnormal growth had come to a standstill 2 years previously. The fact that the condylar fossa was very flat suggested that the enlargement of the condyle may have started very early in childhood. Furthermore, the relatively normal distance between the mandibular canal and the mandibular lower border also suggested that in this case, the increase in the body height had continued rather slowly and was still proceeding, so slowly that the patient was of the opinion that it had stopped 2 years previously. I have also seen this in other cases.

Conclusion. Although the initial appearance of the patient might have suggested the diagnosis of H.H. right away, the clear evaluation of the abnormality clinically as well as on the radiographs finally led to the diagnosis of a hybrid form with very strong predominance of the H.H. growth regulator and very little H.E. influence.

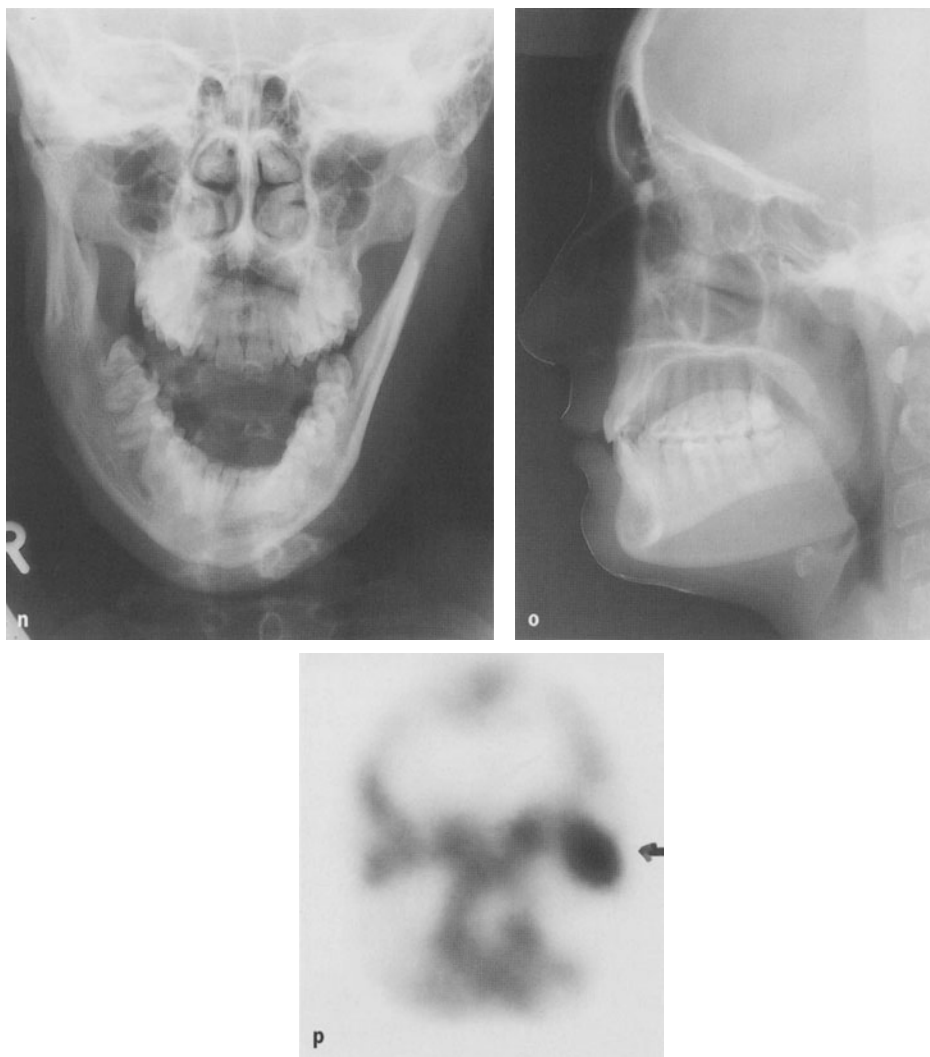


Fig. 66n–p. Unilateral hybrid form. **n** The p.a. projection of the mandible at maximum opening discloses the very severe elongation of the left ascending ramus and the outward rotation of the right hemimandible. **o** The lateral cephalogram shows best the enormous height difference between the two sides. The profile line is not influenced by this. **p** The scintigraphic radiograph shows clear hyperactivity of the condyle on the affected side, compared with the normal side

When the transformation of the coarse trabecular bone structure usually found in H.H. cases extends downwards into the neck and also thickens it, then the diagnosis of a hybrid form of H.E. and H.H. can be made almost on a panoramic radiograph or on TMJ tomograms alone.

Again in this case it was interesting to find that there were highly positive scintigraphic findings in the abnormal condyle in spite of the patient's opinion that the abnormal growth of the mandible had already come to a standstill 2 years previously.

Unilateral Rudimentary Hybrid Form with Clear H.E. Predominance (Fig. 67)

Sex and Age. Male, 20 years of age.

Aetiology and History. The asymmetry had developed gradually for no known reason. It had started at the age of about 12 years and seemed to have stopped 1 year ago. There was nothing similar known in the family history. The patient sought clarification only.

Clinical Findings. The patient's chin asymmetry is very obvious when looking at him from in front (Fig. 67a). The chin prominence is out of the facial midline by about 12 mm to the right side. The left side of the face is longer in the vertical direction than the right with the lower border of the left mandible slightly convex. The lip commissure is slightly oblique, ascending to the shorter right side. Mouth opening is normal, but with less chin deviation than in the closed position. In the profile view (Fig. 67b, c) there is only a slight difference in facial length noticeable on the two sides. The chin

seems rather prominent, but the lips are in correct relationship.

The occlusion picture makes almost everything clear (Fig. 67d–f): There is a class III occlusion on the left side and a class I on the right with a slight crossbite of the cheek teeth in spite of their lingual tilting. All four first bicuspsids are missing. The lower front teeth including the canines are tilted lingually and towards the elongated left teeth. The lower incisor midline is shifted to the right by the width of one incisor. The upper midline corresponds to the facial midline.

Radiographs. The panoramic view (Fig. 67g) shows a strikingly long left condylar neck with a rather small condyle compared with the the right one. Because of this long condylar neck, the left ascending ramus is much longer than the right. Its angle is rounded, while

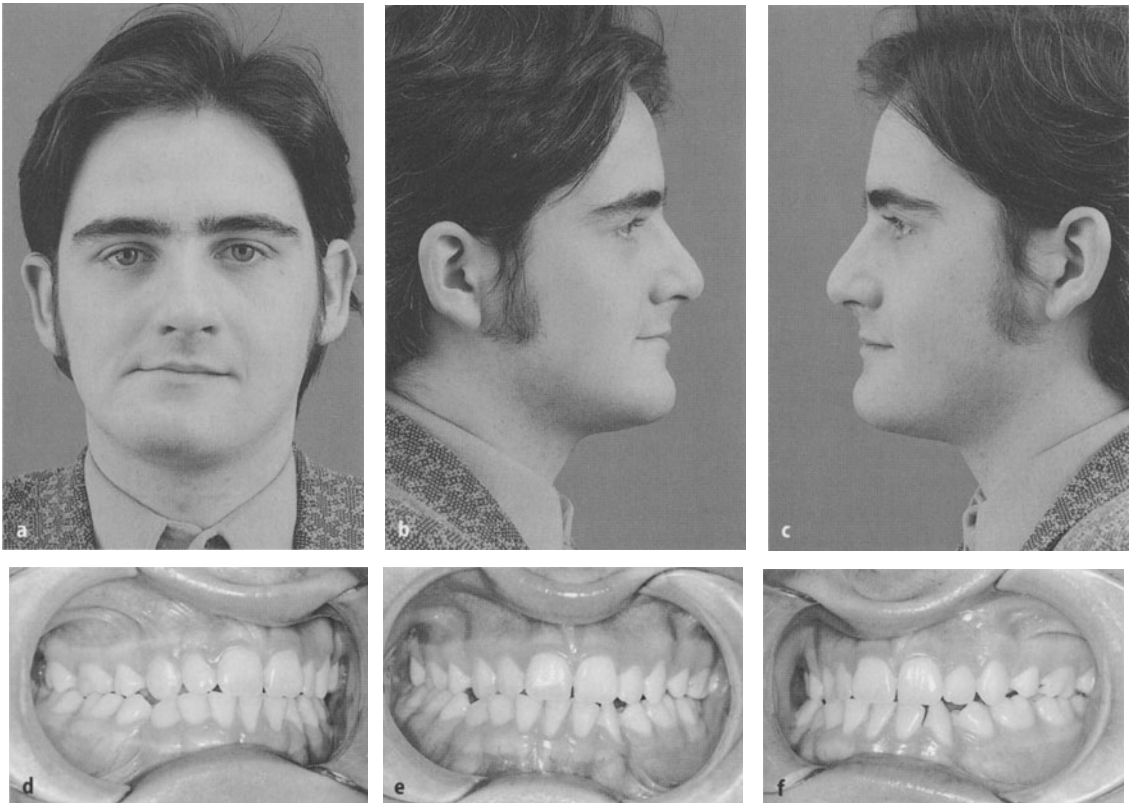


Fig 67a–f. Unilateral rudimentary hybrid form with H.E. predominance. **a–c** The patient's front and profile appearance are very typical of a mild form of a hybrid growth anomaly type with the H.E. part predominant. **d–f** Also the occlusion shows the influence of both growth regulators with the H.E. stronger than the H.H.

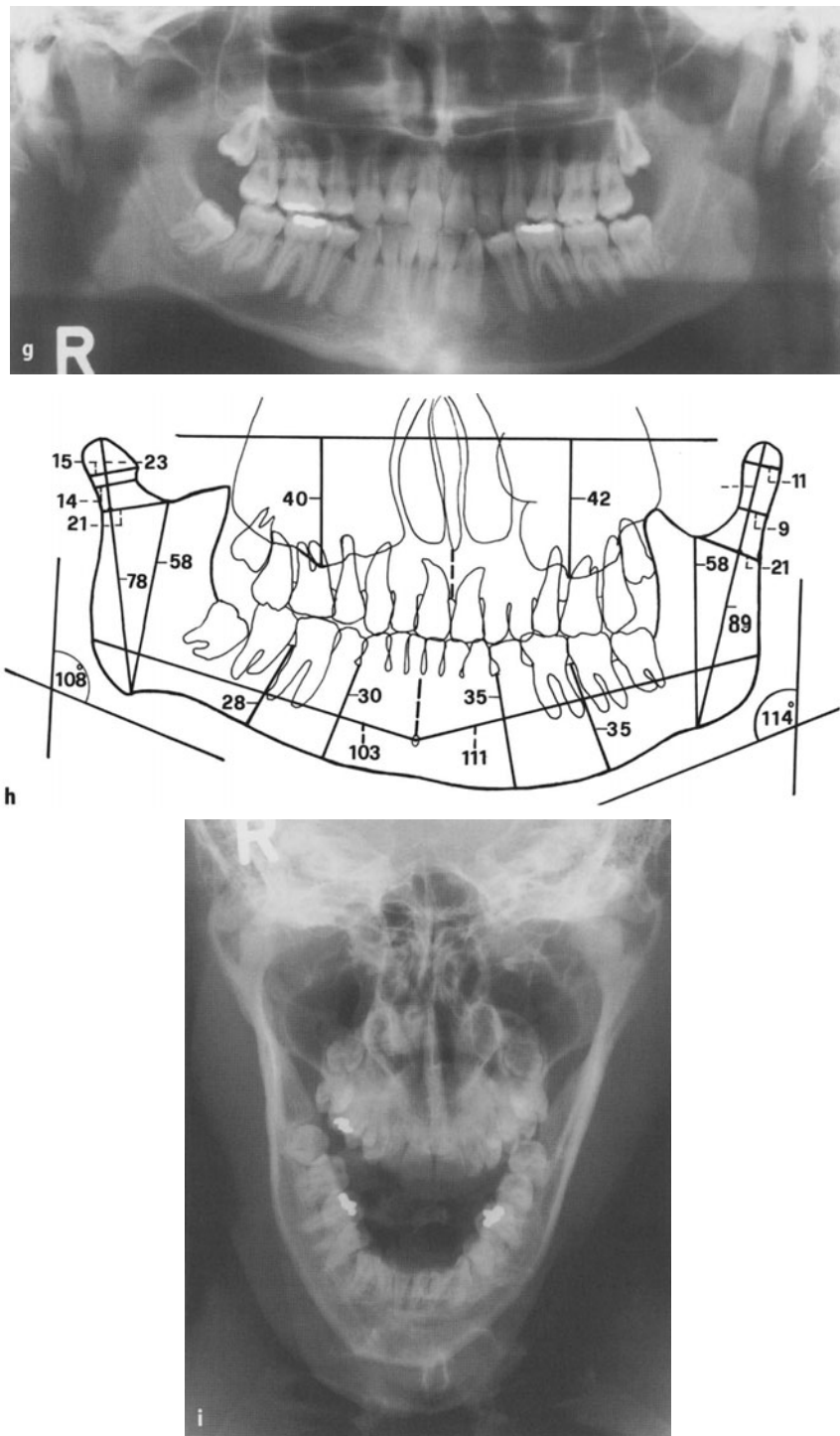


Fig 67g-i. Unilateral rudimentary hybrid form with H.E. predominance. **g** The panoramic view discloses on the left side a long slim condylar neck with a rather small condyle, but with no stretching of the angle. There is an increase in height of the horizontal ramus and a reduction of body height on the right side. **h** The tracing of the panoramic view and its measurements also confirm the diagnosis. **i** The mandible p.a. projection in the open mouth position shows not only a very long left hemimandible but also a condyle clearly larger than that on the right side

the contralateral side has a rather pronounced antegonial notch. The left body has a moderate but clear increase in height in the molar-bicuspid region with a downward convexity, while the right one is rather shallow. The increase in vertical body height reaches as far forward as the symphysis. Besides the rather strong antegonial notch on the right side there is also a reduction in body height in the molar area. But there is a strong ascending ramus and a larger condyle on that side.

TMJ-ramus tomograms could not be taken. The patient refused to have this done.

The measurement of the tracing of the panoramic view (Fig. 67h) shows on the left side, the result of the H.E. influence as well as that of the H.H. and on the right side the secondary consequences: the left horizontal ramus is 8 mm longer than the right and in the molar region 7 mm higher. Both trunks are equal in length but the left ascending ramus is 11 mm longer than the right mainly due to the longer articular process. The Pg-point is out of the facial midline towards the shorter right side by 12 mm. The left angle is well-rounded in spite of the articular process showing the shape of a H.E., slender type.

The mandible in the p.a. projection at maximum mouth opening (Fig. 67i) shows not only the length difference between the ascending rami but also that the left condyle is quite a bit larger than the right. This was not detectable at all on the panoramic view.

Scintigraphy. The patient refused to have this done.

Diagnosis. Unilateral rudimentary hybrid form of H.H. and H.E., very predominantly of the elongation type.

Treatment Plan. The patient refused any surgery. Otherwise a high condylectomy and sagittal splitting procedure on the left ramus were the only suggestions for treatment.

Discussion. This case of a hybrid form of unilateral H.H. and H.E. demonstrated clearly that both condylar

growth regulators can become hyperactive simultaneously and yet with differing intensities. In this case it was mainly the L-regulator which was highly active with the M-regulator much less so. Therefore, the mandible showed mainly elongation signs with a shift to the opposite side. But contrary to the pure elongation form, there was no flat stretched angle but a clearly enlarged rounding of it. In addition, the height increase in the body region with its downward convexity indicated the existence of some degree of H.H. in this mandibular asymmetry.

The shape of the right mandible in the panoramic view was not easy to understand. The rather strong antegonial notch together with the clear reduction in body height in its molar area could suggest damage to the condyle in the growth period. But the strong ascending ramus and the large condyle did not fit that picture. The height reduction in the molar region could have been the result of the pressure from the H.H. component of the other side. I still do not have an explanation for the prominent angle and the antegonial notch. Possibly, good TMJ-ramus tomograms could have been of some value in explaining this matter.

Also, the occlusal situation was very informative. In spite of the lateral position of the mandible with a slight crossbite on the right side, there was noticeable lingual tilting of the right upper and lower cheek teeth. That made them look shorter than the left ones. This is a sign known in unilateral H.H. The severe shifting of the midline to the shorter side is known in H.E. So the diagnosis of a hybrid form of condylar growth anomaly with predominance of the H.E. part can almost be made from the occlusal situation alone.

Conclusion. There is always the possibility that both condylar growth regulators can become hyperactive in the same condyle but with differing intensities. At any one time, the M-regulator may be predominant or the L-regulator, as in this case, or both may be almost equally strong. This case also showed the importance of several different X-ray projections.

Unilateral Active Medium Degree Hybrid Form with H.H. Predominance (Fig. 68)

Sex and Age. Male, 19 years of age.

Aetiology and History. The patient had no idea why his face had become asymmetrical. He said that in childhood the left side of the face was longer than the right. When he was 17 years and 5 months old he was attending our outpatient clinic because of his facial asymmetry. As he believed that the asymmetry was still increasing it was suggested that he wait for 2 more years. In those days we were not of the opinion that high condylectomy should be performed at any stage of a H.H. or of a hybrid form. Scintigraphy was also not then being used in these cases. Exactly 2 years later, his asymmetry had indubitably increased. The left side of the face had become longer and the lower border on that side of the mandible was more inwardly rotated and the lower border on the other side was remarkably more outwardly rotated than 2 years previously.

Clinical Findings. The patient's face has the very typical appearance seen in unilateral H.H. cases with the long, flat face on one side and a rather short one on the opposite side with its angle region more lateral and positioned higher than normal (Fig. 68a–d). The lower border of the longer side has a downward convexity and is tilted medially. The lip commissure is slightly oblique, descending towards the longer side with a downward pull of the corner of the lip as is the occlusal plane. The midline of the prominent chin is over to the shorter side by about 8 mm. The facial innervation and musculature function are normal. Mouth opening is unimpeded, but deviates to the shorter side.

The occlusion at the age of 19 (Fig. 68e–g) is not easy to link with a specific mandibular growth anomaly. On the right side there is a clear class I occlusion and the left side is in a slight class II relationship with a bit of an open bite. But it is clearly visible that the cheek teeth of the shorter right side are tilted slightly lingually and seem shorter than those on the elongated side. But there is no crossbite on the right side.

Radiographs. *The panoramic view* at age 19 (Fig. 68h) and the measurements of its tracing (Fig. 68i) seem to disclose the skeletal background of the asymmetry very clearly. The right ramus is of normal shape and length. The angle of the jaw on that side is normal but there is a height reduction of the body in front of the angle compared with normal and in particular compared with the other side. On that left side, the length of the ascending ramus including condyle and neck measures 18 mm more than on the right side. The left trunk is 6 mm longer than the right. Its posterior border is in an almost vertical straight line. The condyle is large and has

the usual anterior “beak” of a H.H. deformity. The neck is also clearly thicker and longer than normal. In the region of the first molars the height difference in the two bodies measures 9 mm and in the region of the second bicusps it is 5 mm. The symphysis and the Pg-point (whitish spot) is out of the midline towards the shorter right side by 5 mm when compared with the midline of the upper teeth. There is a clear kink at the lower border in the symphysis region. The trabeculation structure is clearly coarser in the body area on the longer left side. The mandibular canal is easily visible. It runs parallel to the lower border at a distance of about 2–3 mm, on the higher side, a bit further away than on the right. The lower front teeth are tilted to the longer side.

The TMJ–ramus tomograms (Fig. 68j,k) show a normal shape and size of the ascending ramus, condyle and neck on the right side. The left side confirms what had already been seen on the panoramic view. There is a much enlarged condyle with an irregular surface and an eagle-like “beak” on the anterior part. Its neck is clearly thickened and its coarse trabeculation extends down into the neck.

The mandible in the p.a. projection at maximum mouth opening (Fig. 68l) shows clearly the elongation of the left ramus with the enlargement of the condyle and the latter's coarse trabeculation extending down into the upper half of the neck. It cannot show the vertical height increase in the body. The medial tilting of the whole left hemimandible and the outward rotation of the right side is clearly visible and also the two halves of the chin prominence with the left side further anterior than the normal right side. This proves that the whole left hemimandible is involved in the condylar growth hyperactivity.

The lateral cephalogram showed clearly the deep overbite and the vertical height difference between the two sides.

The p.a. cephalogram (Fig. 68m) shows not only the height difference between the two sides but also very clearly the outward rotation of the shorter right body and ramus and the inward tilting of the long left mandible. The coarser trabeculation is also clearly visible in the left ramus. This projection also proves that the vertical height increase in the right side of the maxilla is the cause of the oblique occlusal plane.

The symmetry evaluation on the tracing of this p.a. cephalogram makes everything clear (Fig. 68n) about the skeletal situation. There is not only an increase of vertical dimension of the affected side. The Sy-point too is clearly over to the non-affected side, indicating an increase of horizontal dimension on the affected side.

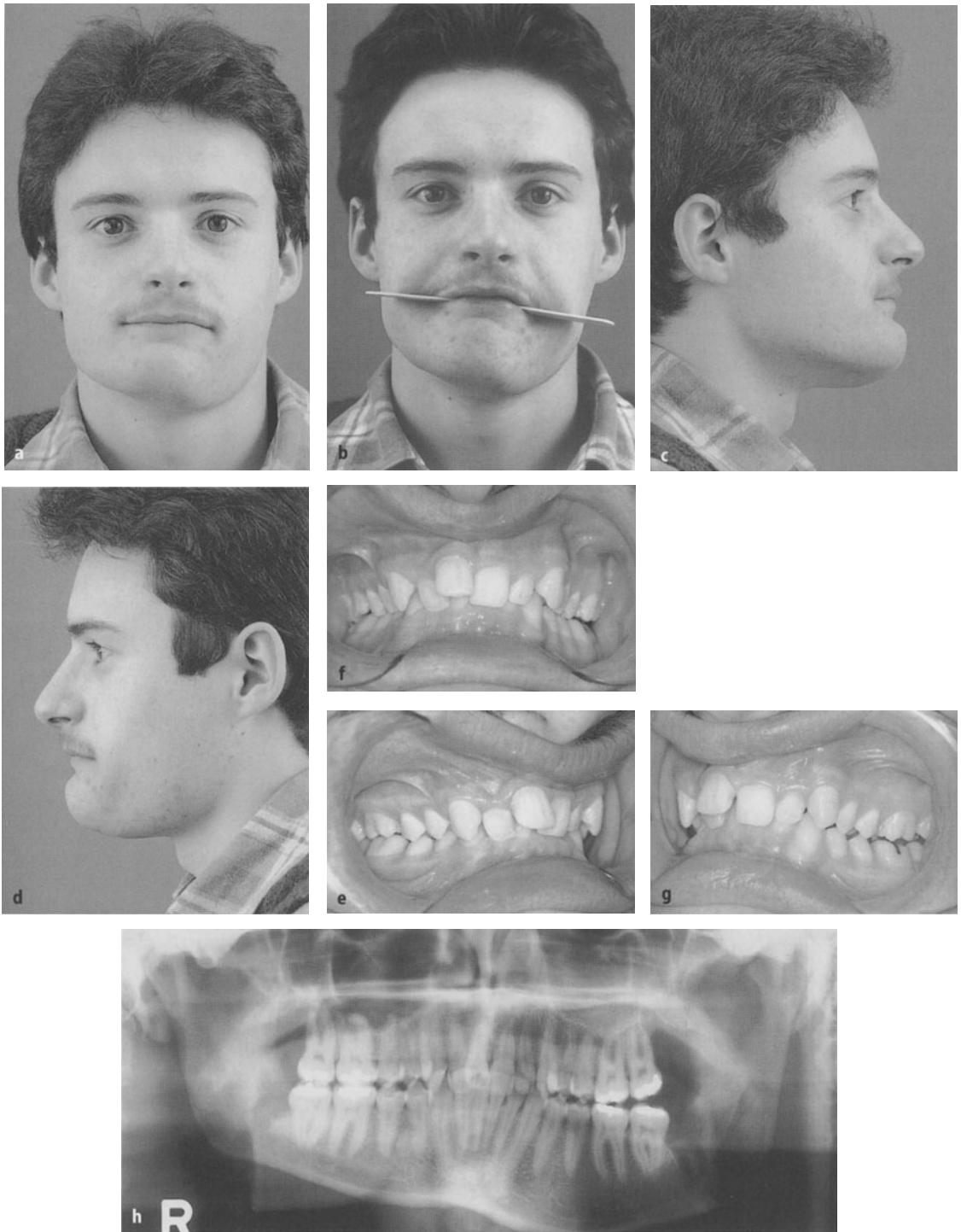


Fig. 68a–h. Unilateral active medium degree, questionable hybrid form, with strong H.H. predominance. **a–d** The patient's appearance at age 19. **e–g** The patient's occlusion at age 19 typical of a H.H. anomaly, but no crossbite. **h** The panoramic view at age 19 shows the complete signs of a left side H.H., but also the symphysis out of the midline to the shorter right side

Provisional Diagnosis. Unilateral questionable hybrid form of H.H. and H.E. with the H.H. component very predominant.

Treatment Plan. Left intracapsular high condylectomy and resection of the surplus of the lower border using the resected piece of bone as an onlay graft on the same side, because of its medial displacement.

Actual Treatment. This was carried out by H. Sailer, then senior coworker at my clinic. After high condylectomy via a retrotragal approach, the mandibular nerve in the body of the mandible was freed via an intraoral approach to above the angle. Then the lower surplus was cut off with a saw and fixed laterally to the new lower

part of the body. A perfect aesthetic result was achieved. The patient refused any orthodontic treatment.

Histology. Macroscopically, the resected specimen appeared enlarged and distorted. Microscopically, the bone towards the articular surface was covered entirely by slightly thickened hypertrophic growth cartilage which, however, was partly cut off at one end of the specimen. In the same area, the otherwise clearly evident proliferative layer was missing, whereas the articular layer was visible only along a relatively small part of the articular surface at the opposite end of the condyle (Fig. 68o). Due to poor tissue preservation, only a few large cells resembling chondroclasts and a few seams of osteoblasts could be recognized at the lower limit of the

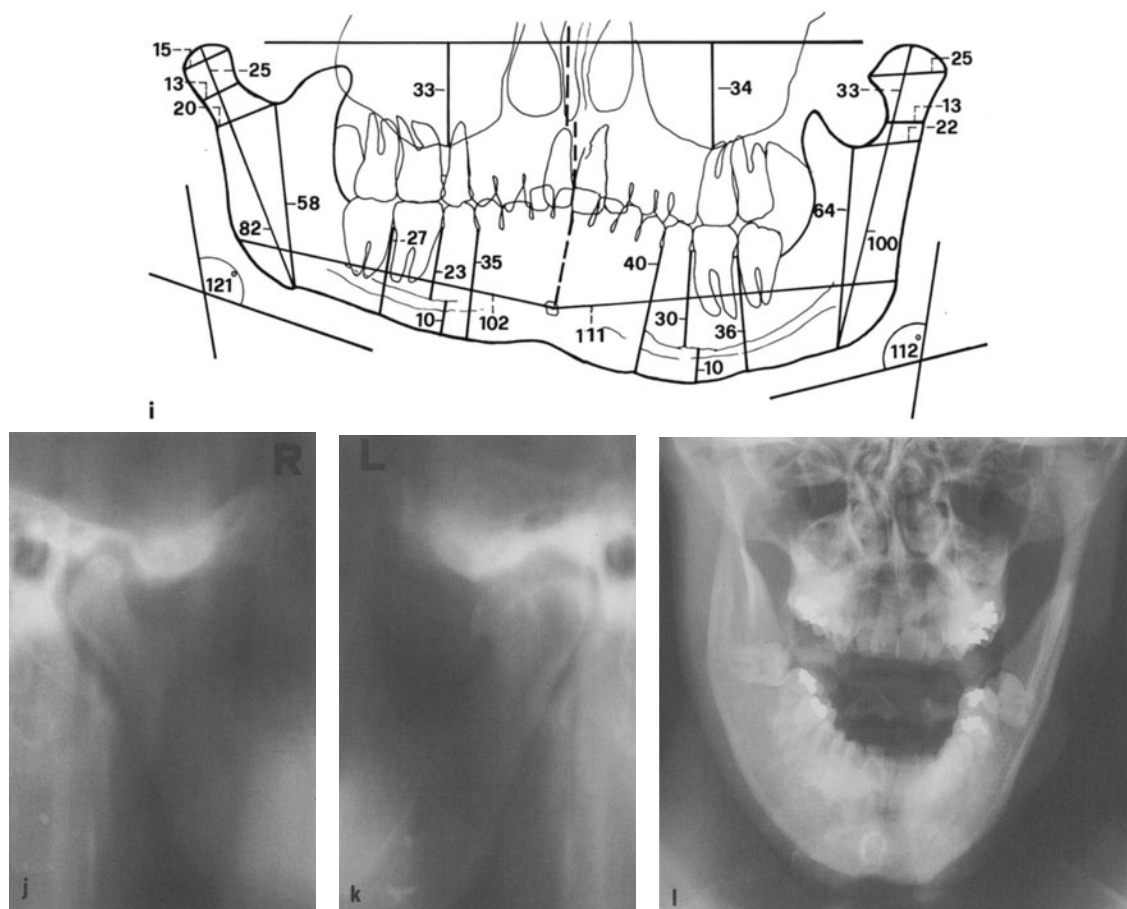


Fig. 68i-l. Unilateral active medium degree, questionable hybrid form, with strong H.H. predominance. **i** The measurements of the tracing seem to support the diagnosis of a hybrid form with strong predominance of the H.H. part. **j, k** The TMJ ramus tomograms show a normal right condyle-ramus situation and a very enlarged left condyle with a long and rather thick neck. **l** The mandible p.a. projection at maximum mouth opening shows all the requisite signs of a H.H. anomaly with a very much elongated left hemimandible. Its condyle is large and it has a long neck with coarse trabeculation

hypertrophic cartilage. Yet, absence of a subchondral bone plate and, instead, the presence of extensive marrow spaces in direct contact with the cartilage (Fig. 68o) and cartilage rests down to the resection border indicated highly active endochondral ossification which undoubtedly exceeded that observed in age-matched controls (Fig. 68p). Also in contrast to these control specimens, trabeculae of the subchondral spongiosa were

thin, ill-oriented, and arranged in a tight network around small marrow spaces (see Fig. 79h).

Diagnosis. Malformed condyle exhibiting excessive late growth and a dysplastic spongiosa characterized by a tight network of haphazardly arranged trabeculae containing extensive cartilage islands.

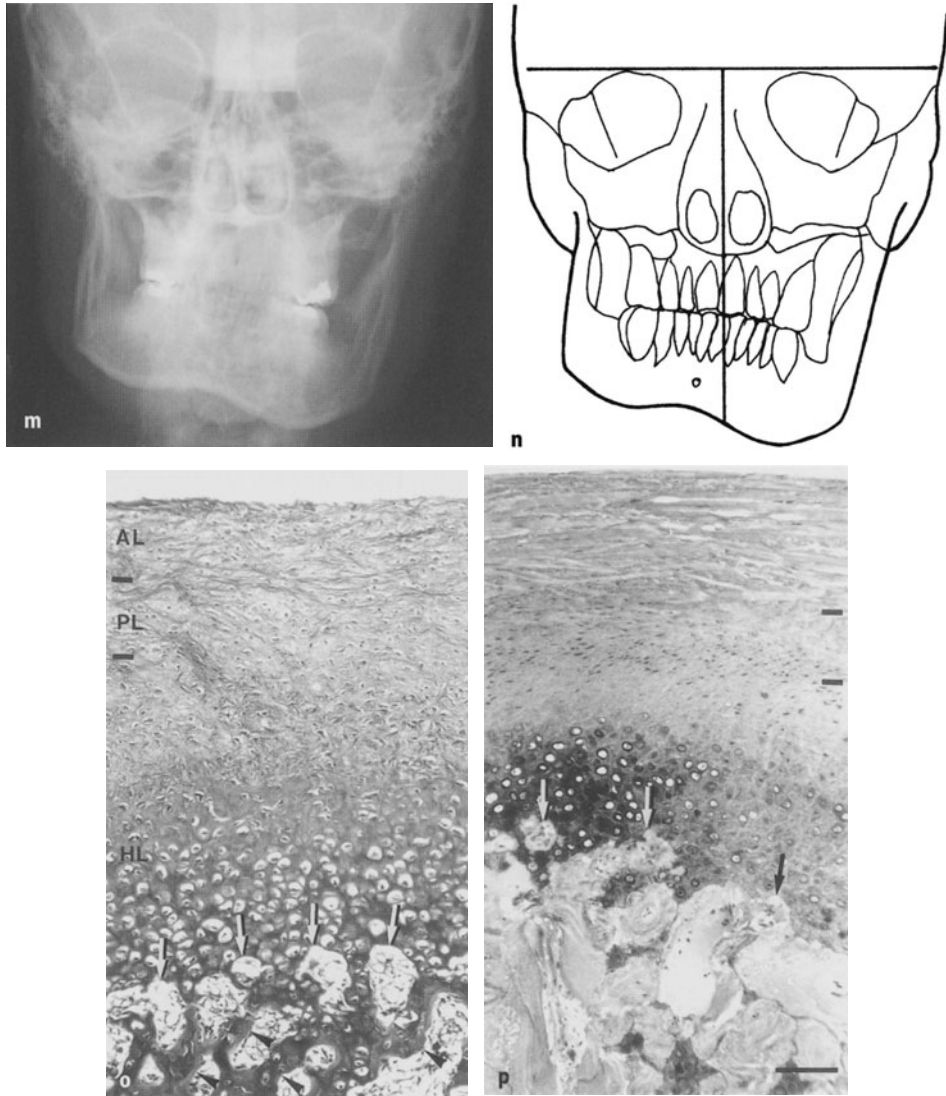


Fig. 68m–o. Unilateral active medium degree, questionable hybrid form, with strong H.H. predominance. **m** The p.a. cephalogram also shows clearly the enlargement of the left side of the mandible with its coarse trabeculation and the symphysis over to the right side. It also shows a height increase in the left side of the maxilla. **n** The tracing of this p.a. cephalogram makes it clear that there is also a degree of H.E. influence in the anomaly in spite of the absence of a crossbite. **o, p** Microscopic appearance of the articular tissue of the excised condyle **o** in comparison with that seen in a normal specimen also from a male of about 20 years of age **p**. Note the thick, clearly hypertrophic cartilage (**HL**), the numerous lacunae (*arrows*) in the zone of cartilage erosion and the tight network of trabeculae (*arrowheads*) in the primary spongiosa of the affected specimen **o**. In the normal condyle **p**, cartilage matrix is comparatively abundant at the expense of chondrocyte size and the subchondral bone forms a fairly continuous plate (**AL** articular layer, **PL** proliferative layer. **o** paraffin section, **p** plastic section, toluidine blue, original magnification $\times 130$, bar = 100 μ m)

Comment: These observations, in particular those regarding the subchondral spongiosa, closely resemble those made in a case of H.H. (see Figs. 37cc, 79b). Thus, the histological appearance of the condyle, lacking any sign of the H.E. component, would appear to largely reflect the clinical picture that is characterized by the predominance of H.H.

Discussion. A case may initially look like a straight forward H.H. or a H.E. anomaly. But there are several points which should be considered to be sure of the diagnosis. An important one is the position of the chin prominence. In a pure H.H. the chin prominence as a whole will not be over to the opposite side, except when the contralateral side is a hemimandibular hypoplasia. When one is in doubt about the position of the symphysis, which equates to the anatomical middle of the chin prominence, then draw a horizontal line above the eyebrows and to it a perpendicular line from the middle of the forehead down beyond the chin region. One can do this on the patient's face or on an enlarged photograph or on radiographs.

Another point is the fact that a pure H.H. anomaly will always show a bilateral chin prominence effect with the H.H. side more prominent. This is the case here too. The half on the anomaly side will be more prominent than the other. If there is also some H.E. hyperactivity involved on the same side, then that eminence difference may be less obvious and in a case of approximately equal influence of both growth regulators, the difference will almost disappear.

Another important point is the condylar neck. In a pure H.H. case it is the condyle only which is enlarged while the neck remains nearly unchanged. When the condylar neck is clearly elongated then a H.E. influence may exist. This is particularly true when the neck is longer and remarkably thicker than normal, as in this case. The greater the percentage of H.E. influence, the more the chin and the occlusion will be shifted over to the opposite side.

But there was one important sign of H.E. missing in this case. That was the crossbite. So where did the truth lie? Going back to the panoramic view looking for the pogonion and its relation to the facial midline, then we found that I had been misled by the midline of the upper and lower incisors. They were out of the facial midline to the left side by about 5 mm, clearly seen in their relation to the straight nasal septum. That meant that there was no clear sign remaining of a H.E. influence in this growth anomaly. But what when the nasal septum is out of the facial midline? In that case one may make a wrong diagnosis. In order to be sure about the facial midline and the position of the chin and the tooth midline in relation to that facial midline we have to find the position of the crista galli. That will be clearly seen

on the p.a. cephalogram. If one connects the supraorbital rims with a line and draws to it a perpendicular line from the crista galli downwards beyond the chin, then one cannot be misled. This proof is even better than that on the enlarged photograph. It disclosed very clearly that the whole masticatory skeletal complex was rotated to the right side, the maxilla and the ascending ramus on the left side clearly longer than on the right side. The nasal septum was out of facial midline to the right and so was the midline of the upper and the lower teeth, contrary to the findings on the tracing of the panoramic radiograph. Even more out of the facial midline to the shorter right side was the prominence of the chin and the Pg-point. So finally, there seemed to be no doubt about the existence of a hybrid form in this case, with the H.H. very predominant.

But what about the crossbite which we want to see as a specific sign of the H.E. influence? The answer is that the downward rotation on the left side has shifted the whole masticatory complex over to the right side, and by this compensated for the crossbite which would have appeared without that rotation of the lower half of the facial skeleton. So finally, one must confess that it is a case of a hybrid form with the H.H. component very predominant.

The histological picture seems to show mainly the H.H. abnormality but not the existence of an additional slight H.E. component.

Conclusion. When in a case of an apparently pure H.H. the chin prominence is over to the opposite side, then a H.E. influence must also exist if a contralateral hemimandibular hypoplasia can be excluded. In such a case, the upper half of the condylar neck will also be thickened and elongated and will show a structure similar to the enlarged condyle.

But in this case, the neck of the articular process was not more elongated than one could have expected in a very pure H.H. case. And even more important is that the upper half of the neck reveals no signs of involvement as clearly seen in Figs. 66 and 69. Another very important sign of the H.E. influence is the crossbite due to the shift of the mandible over to the other side. This is clearly missing in this case. The conclusion is that the diagnosis of the existence of a pure or hybrid form of growth anomaly in the case in question is not always that easy. It is not important for the treatment. It is mainly an academic but very interesting question. In this case it is noticeable again that the mandibular canal can remain away from the lower border when the condylar M-regulator hyperactivity continues on for many years. In this particular patient it must have already started at the age of 6 years or even earlier and was still active when he was 20 years old.

Unilateral Highly Active Hybrid Form During Puberty (Fig. 69)

Sex and Age. Male, 16 years and 1 month.

Aetiology and History. When the boy was 10 years old his mother recognized for the first time that there was a beginning asymmetry of his mandible, as the chin had moved over slightly to the right side. At the age of 15 years an orthodontist was consulted. He commenced fixed orthodontic appliance treatment to arrange the teeth into such a position that they would fit together when the mandible was corrected surgically. He was referred with the orthodontic appliances in situ for diagnosis and treatment planning.

Clinical Findings. At the age of 16 years and 1 month, the patient is referred with a long, distorted face (Fig. 69a–e). The prominence of the chin is out of the midline to the right by 10 mm. The left side of the face is quite a bit longer than the right. The lower border of the mandible seems twisted outwards on the right side and medially on the longer left side. The left mandible is much longer than the right and has a downward convexity of its lower border. There is a slightly oblique occlusal plane. Mouth opening is normal but with a slight deviation to the shorter right side.

The teeth have orthodontic brackets and bars in situ. There is a flat open bite on the left side and in the anterior region (Fig. 69f). The lower incisor midline is shifted over to the right by the width of 1 2/3 lower incisors. There is a crossbite on the shorter right side. The maxilla seems a bit higher on the longer left side. Impressions of the teeth were taken. The models so obtained could not be brought into proper occlusion. The patient's orthodontist said it would take him a few more months before the dental arches would be ready for surgery.

Nine months later the boy, now 16 years and 10 months old, was referred for surgery. Since he was last seen by us his deformity has increased dramatically (Fig. 69h, i). The asymmetry has become much worse. The chin is further over to the right by an additional 10 mm. The lip commissure has become very oblique, descending to the abnormal left side. The discrepancy in length of the two hemimandibles is now even more impressive, particularly visible when looking at the patient's face from above. Mouth opening is unimpaired, almost correcting the chin asymmetry. The occlusal situation has also changed very impressively (Fig. 69g). The open bite has moved from the left to the right side and has increased. The midline of the lower incisors is now over to the normal right side by the full width of two lower incisors. The negative overjet and the lateralization of the mandible to the right has increased considerably. The models of the two dental arches could now be brought together very nicely.

Radiographs. The hand radiograph showed almost perfect complete closure of the epiphyseal plates of the fingers and the radius and ulna at the boy's age of 15 years and 1 month.

The panoramic view (Fig. 69j) taken by the patient's orthodontist at the same age in a slightly open position, shows, at first glance, an almost normal mandible, but with an elongated left body, a large condyle with a thick neck and the lower incisor midline is over to the right side by the width of a little more than one incisor. The front teeth are tilted towards the longer left side as in an unilateral H.E. case. The mandibular body on the left side is slightly higher in the bicuspid-molar region than on the right side and it is slightly convex. The roots of the bicusps and of the first molar are separated more than normal and than seen on the right side, obviously due to additional alveolar bone production. The cortex of the lower border on the abnormal side is very thin, almost translucent and trabeculation is almost non-existent in that premolar region. In that whole area the radiological structure of the body looks less distinct. The left angle is much more rounded with a larger radius than the right. The left ascending ramus looks heavy and has a very much enlarged condyle with a "beak" at its anterior surface. Its neck is almost double the thickness of the right one which is of normal shape and thickness. This is also true for the right condyle.

The lateral cephalogram taken at the same age disclosed the already severe asymmetry with a downward convexity of one mandibular body and a negative overjet.

The panoramic view taken exactly 1 year later (Fig. 69k), at the boy's age of 16 years and 1 month, now shows all teeth with the fixed orthodontic appliance in place and a clear open bite in the front, mainly in the region from the right lower canine to the left second molar. The midline of the lower teeth is now shifted to the right side by the width of two incisors and the lower front teeth are tilted to the long left side. The mandibular canal is not recognizable in the body region. The downward convexity in the left premolar region has increased slightly. There is now a remarkable height difference of 10 mm between the left and right maxilla.

We know that the measurements of two panoramic view projections are not comparable. The striking reduction of the postero-anterior width of the two ascending rami and of the condylar necks may well be a product of different projections as the whole length of the mandible from one G-point to the other is 40 mm longer on the first radiograph than on the second. That the width of the condylar neck is 3 mm greater in the

first radiograph must surely also be due to the same technical reason. In spite of that fact some findings may provide valuable information.

Regrettably, no new panoramic view was taken immediately before surgery after Fig. 69k.

The measurements of the tracing of the panoramic radiograph at 16 years and 1 month (Fig. 69l) reveals clearly the differences between all sections of the two hemimandibles and the two sides of the maxilla. The left side of the maxilla must have rapidly followed the inferior downward growth of the mandible, probably already before puberty. The main differences in length are between the horizontal rami (24 mm) and the ascending rami (14 mm). While the difference between the trunks measures 6 mm only, it is clearly the difference

between the two articular processes (14 mm) which contributes to the major part of the length difference of the two ascending rami. That means that all sections – the horizontal ramus, the trunk and the articular process of the left hemimandible – are elongated when compared with the normal side. This is almost pathognomonic for the H.E. anomaly. As far as the H.H. part is concerned in this hybrid case we would also like to see that the same sections have increased in mass. This is definitely true for the articular process and the horizontal ramus. This panoramic radiograph does not permit evaluation of the trunk from that point of view, neither in its breadth nor in its thickness.

Looking at the p.a. projection of the mandible in the closed position before surgery (Fig. 69n) and 1 year af-

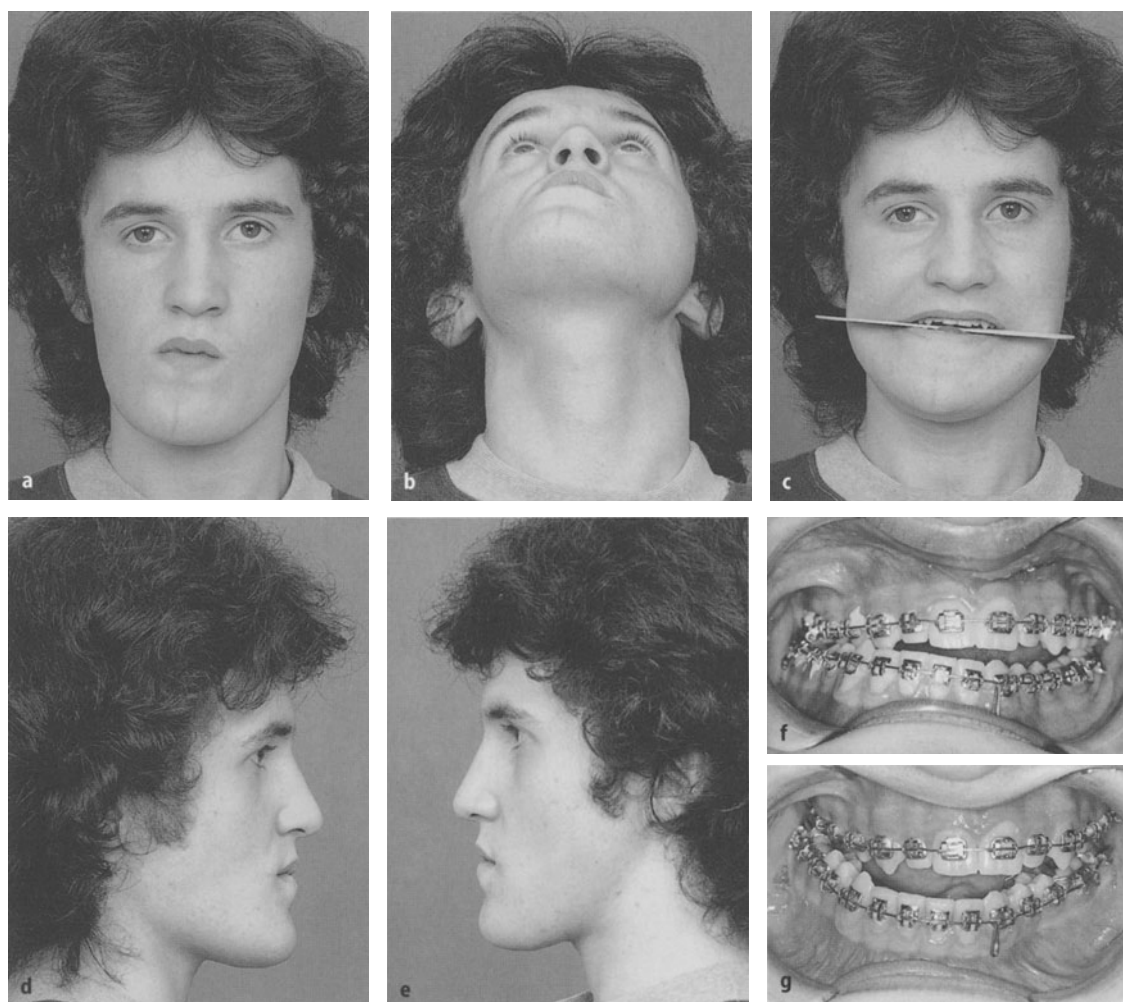


Fig. 69a–g. Unilateral highly active hybrid form during puberty. **a–e** The patient's appearance at age of 16 years and 1 month presenting severe facial deformity and an oblique occlusal plane. **f** The patient's occlusion at the same age: forward and lateral position of the mandible with a flat open bite, mainly on the left side. **g** Nine months later the open bite has increased and changed over to be mainly on the right side

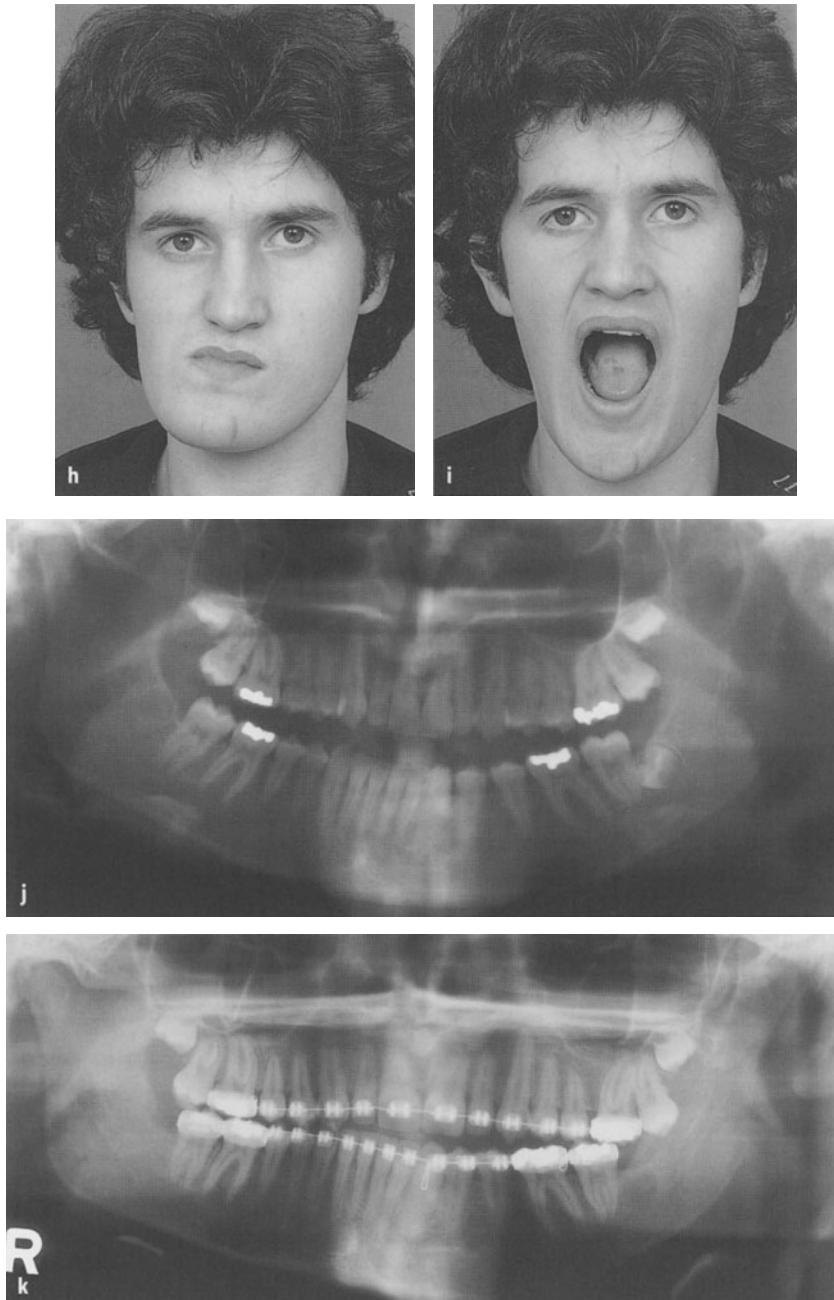


Fig. 69h–k. Unilateral highly active hybrid form during puberty. **h, i** The patient's facial deformity has worsened almost dramatically within 9 months. At maximum mouth opening there is not much of the severe facial asymmetry noticeable. **j** The panoramic view, taken at age 15 years and 1 month, discloses a rather long left hemimandible half with a large condyle and wide neck together with a slight increase in the left body height. **k** The panoramic view, taken 1 year later at 16 years and 1 month, reveals the amount of change in the mandible within 12 months

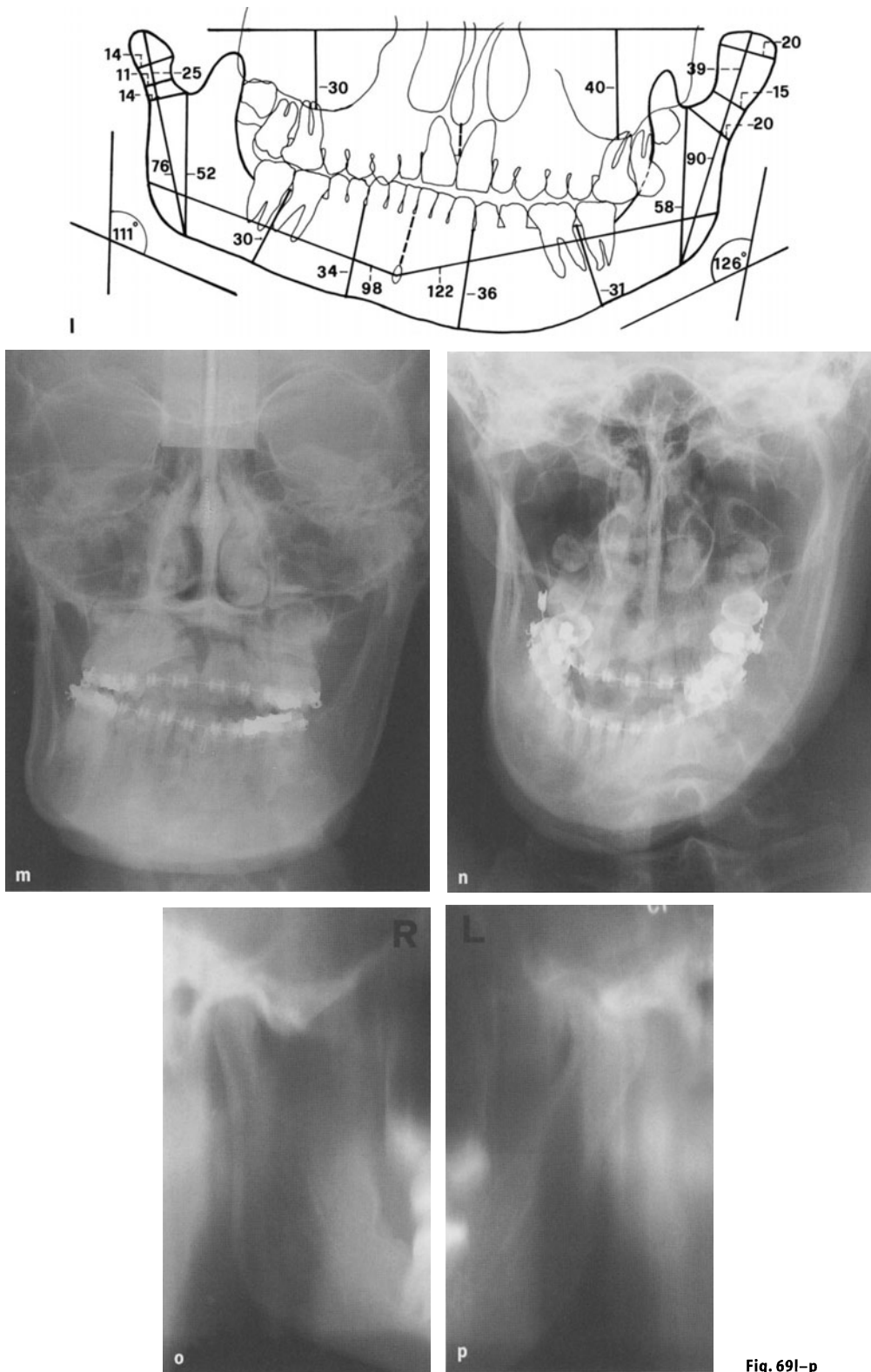


Fig. 69l-p

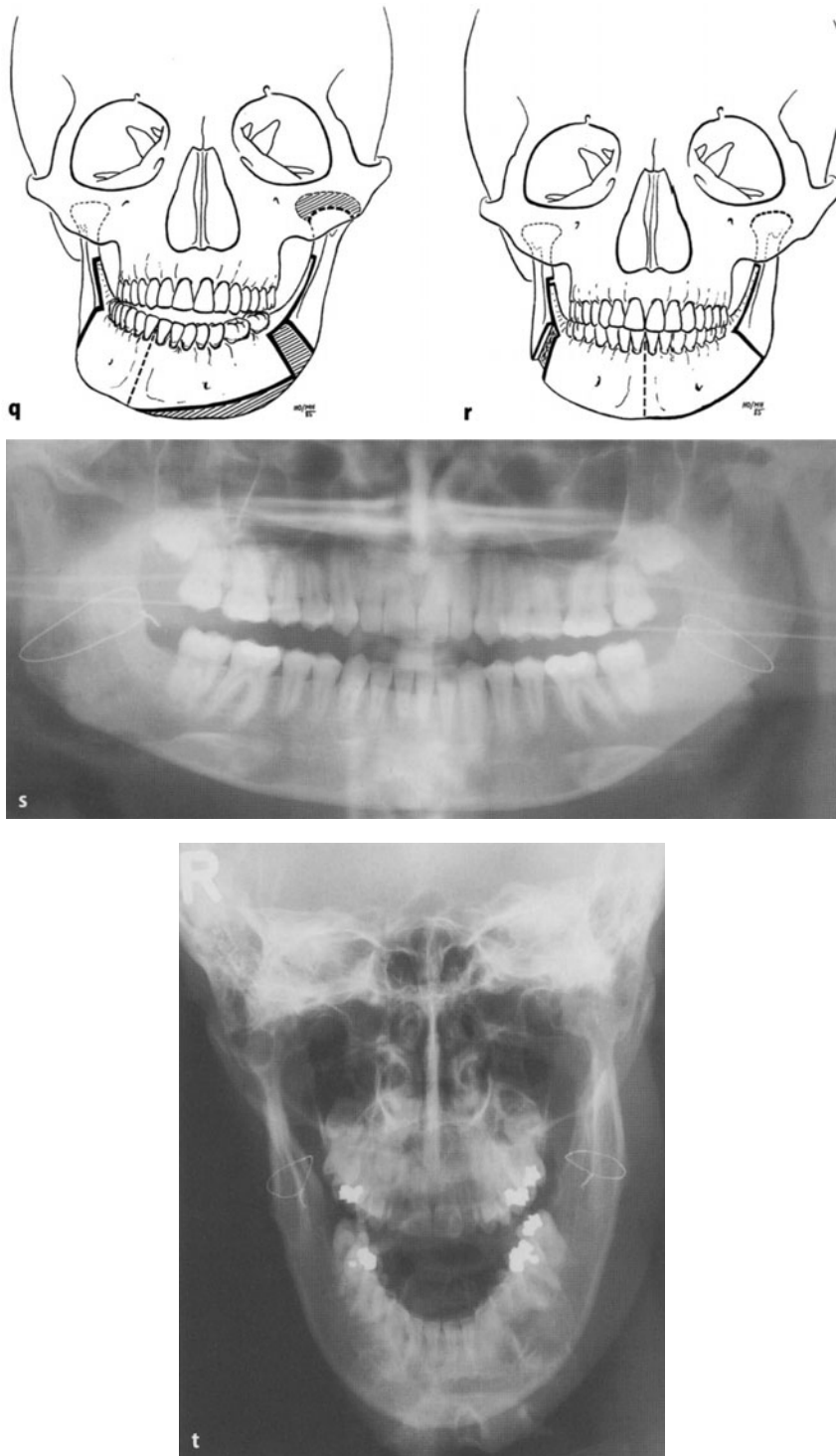


Fig. 69l–t. Unilateral highly active hybrid form during puberty. **l** The tracing of the panoramic view of **Fig. 69k** reveals very clearly the differences between left and right, in length as well as in mass, of all sections of the mandible. **m, n** The p.a. projection of the mandible in the closed position, taken only 9 months apart, demonstrates the enormous surplus of growth of the mandible in such a short time. **o, p** The TMJ ramus tomograms show a normal right and a very deformed left condyle-ramus situation. **q, r** Diagrammatic illustration of the planned corrective surgery. **s, t** The panoramic view and the mandible p.a.-projection in the open mouth position, taken 1 year after surgery, disclose that the mandible has become nearly symmetrical

ter surgery in the open mouth position (Fig. 69t) confirms the conclusion that the left ascending ramus is clearly thicker than the right. With that fact there is proof that this hemimandible is, in all its sections, not only longer but has also a surplus in volume or mass of bone.

Comparison of the *p.a. projection of the mandible* in closed position taken at the age of 16 years and 1 month (Fig. 69m) with the same projection *taken 9 months later* (Fig. 69n), shows the tremendous change in the mandible within such a short time. Holding the facial midline of both radiographs in a vertical position one is impressed not only by the enormous increase in length of the left ascending ramus but also in the length of the whole left side of the mandible. It represents an overgrowth in length and mass of the left mandible, obviously followed by a downward growth of the maxilla on the left side. The open bite is now located more to the right side than 9 months previously, when it was more on the left side. The *p.a. projection of the mandible* in the closed position before surgery (Fig. 69n) shows the dramatic asymmetry of the lower half of the face even better than does the panoramic view, which was taken 9 months earlier (Fig. 69k).

The *TMJ-ramus tomograms* taken at the age of 16 years and 8 months (Fig. 69o, p) show a normal right ra-

mus, condyle and neck with a well-formed angle on that side of the mandible. The left side tomogram (Fig. 69p) shows that the angle is almost straight and there is a very thick condylar neck and very large condyle with a rather thick, upwardly directed "beak" in its anterior region. That "beak" articulates with the base of the skull in front of the articular eminence and has formed an additional glenoid fossa-like concavity.

Regrettably, *scintigraphy* was not requested.

Provisional Diagnosis. Highly hyperactive unilateral hybrid form of H.H. and H.E. due to almost equal influence of the two condylar growth factors during puberty.

Treatment Plan. After finishing the presurgical orthodontics impressions were taken for the model operation. It proved that the patient's orthodontist, Dr. Bolter from Wädenswil, had done an excellent job as a perfect occlusion could be achieved. The plan for surgery then was intracapsular high resection of the left condyle and a bilateral sagittal splitting procedure on the rami and if necessary additional sliding correction of the chin prominence as well as reduction of the slightly excessive height of the left mandibular body (Fig. 69q, r). An addi-

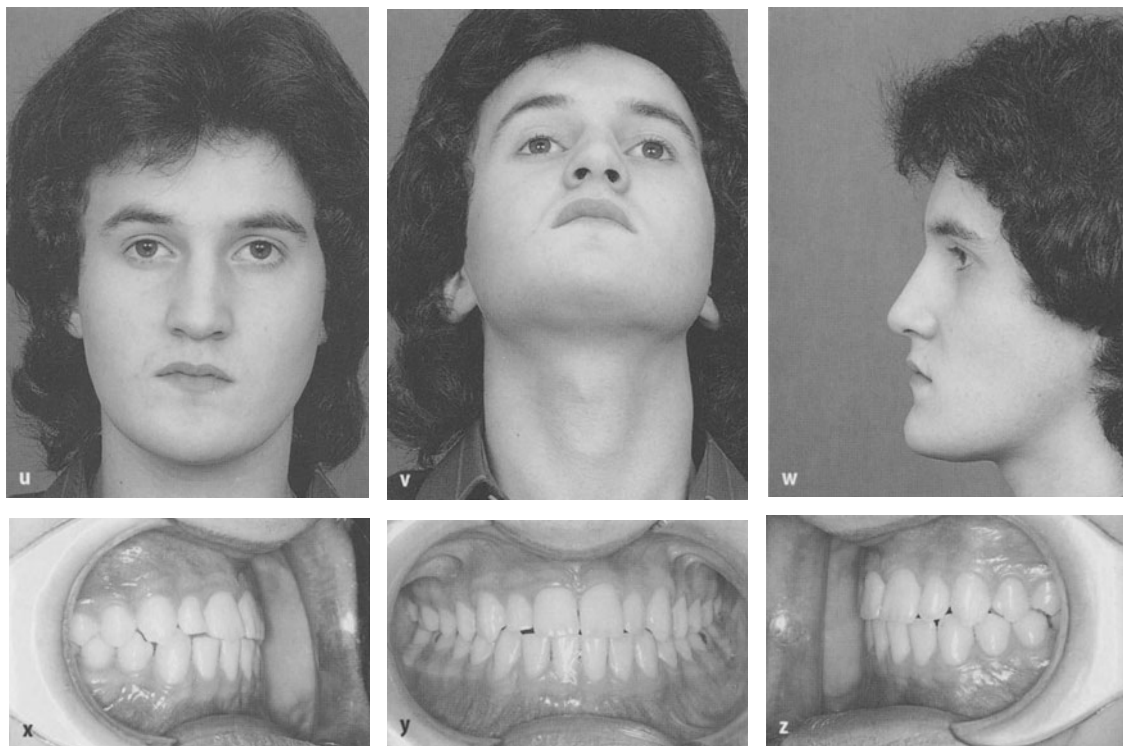


Fig. 69u-z. Unilateral highly active hybrid form during puberty. **u-w** The patient's appearance 1 year after surgical correction **x-z** The patient's occlusion 1 year after surgery

tional rotation of the lower half of the facial skeleton was found not to be necessary, regrettably.

Actual Treatment and Result. A very experienced trainee was asked to perform the operation in accordance to the plan. First, the upper half of the condyle was resected. Next, the sagittal splitting of both ascending rami was performed. After the mandible was completely mobile, it could be fixed to the upper jaw in the planned occlusion. Following intermaxillary fixation the overlapping part of the left proximal segment was resected and a circumferential wire on each side guaranteed good adaptation of the segments in the new position. As the shape of the mandible and also the face seemed to be almost symmetrical, the surgeon decided that there was no need to move the chin prominence or remove the slight convexity of the left lower border. It is recorded that the resected upper half of the condyle had "beaks" in addition to its irregular enlargement. The intermaxillary fixation was left for 6 weeks.

One year after surgery, radiologically (Fig. 69s, t) a rather nice symmetry of the mandible has been achieved.

The patient's appearance and occlusion photographs (Fig. 69u-w) show the satisfactory clinical result. However, there is still a slightly longer left side of the face and an oblique lip commissure as a consequence of not rotating the whole lower half of the face including the maxilla, and not reducing the surplus of height of the left body with its inferior convexity.

Histology. The micrographs taken earlier from the resected condyle disclosed considerable soft tissue damage that seemed to have been due to surgical injury or histological processing. Yet a thick layer of hypertrophic growth cartilage could be clearly recognized. Numerous marrow spaces in direct contact with the cartilage and abundant cartilage islands embedded in the subchondral bone trabeculae suggested active endochondral ossification. Compared with those seen in the previous case (Fig. 68o), a hybrid form with predominance of the H.H. component, these trabeculae were most evidently thicker, less numerous, and better oriented.

Diagnosis. Actively growing, structurally inconspicuous condyle.

Comment: Unfortunately, no sections were available and histological examination had to rely entirely on old photomicrographs. The inconspicuous condylar appearance revealed by these photomicrographs seems to be compatible with the clinical picture of a hybrid case

without a predominance of either the H.H. or the H.E. component. In contrast, the clearly enhanced growth of the affected condyle recorded clinically is not apparently reflected by the structure of either the cartilage or the subchondral spongiosa.

Discussion. This case of a hybrid form of H.H. and H.E. had started at the age of about 10 years. It became almost explosive during presurgical orthodontics between 15 years and 1 month and 16 years and 10 months, although the general skeletal growth had practically finished at the age of 15 years according to the hand radiograph. The shape of the mandible surely suggests that the H.E. was slightly predominant in this case, but on the panoramic radiograph only. H.E. alone could never produce such a rotated face. The H.E. shifts the mandible over to the other side but does not produce such a voluminous jaw. With the vertical height difference, this case was clearly a hybrid form, in spite of the fact that the vertical height increase in the body was rather mild. Also, the shape and size of the condyle and its neck was very typical and almost pathognomonic for hybrid hyperactivity. H.E. would not produce such a condyle and neck deformity. Although it is very interesting to follow the development of this type and severity of mandibular asymmetry, for the patient it would be best to do a high condylectomy as soon as the development of a hybrid form can be confirmed.

It is really a great pity that in this dramatic case of misregulation of mandibular growth, only photomicrographs were available for histological examination and not the specimen itself. It does not seem to make sense that such important parts of a patient's records such as radiographs or histological specimens should be destroyed after 10 or 20 years. What a pity!

Conclusion. This case proves that in the hybrid form of H.H. and H.E. surplus length as well as of volume or mass of bone is found in all sections of that hemimandible. Depending on which of the two growth regulators is predominant and to what extent, the amount of overgrowth will be determined. In this case it was clearly the influence of the L-growth regulator. This case also proves that a hybrid form of H.H. and H.E. seems to be able to develop in an almost explosion-like manner and with such severity that is not experienced in the pure H.H. or H.E. forms. Therefore, high intracapsular condylectomy must be advocated as soon as one can diagnose the beginning of a developing hybrid form, this in spite of the fact that there are also mild forms which may finally not even require surgery.

Bilateral Asymmetrical Questionable Hybrid Form with Relapse after Surgical Correction (Fig. 70)

Sex and Age. Male, 22 years and 7 months.

Aetiology and History. The patient had no idea why his lower jaw had grown forwards more than normal. He did not know when the forward growth started, but when he was 16 he already had a very protruding mandible. At that time it was even worse than now, he said. His growth had finished when he was 17. Since then he had always taken the same size in shoes and had worn the same signet ring, which he can easily remove, on his finger. The father's mother also had a severely protruding mandible, but he reported no one else in the family history had this.

Clinical Findings. Viewed from in front the patient seems to have a strong facial skeleton with a very prominent and wide asymmetric mandible (Fig. 70a). His supraorbital rims are strong, but his infraorbital rims and the zygomas are rather small and the mid-face area flat. The nose and the ears are small and his skin is smooth. The mandible seems long and asymmetric, protruding in front of the upper lip. The chin is very strong with its prominence out of the midline to the left side by 1 cm. In profile view the face has an acromegalic appearance, with a strong forehead and a protruding mandible, but with a severe dish-face deformity (Fig. 70b).

The mouth opening was normal. His tongue did not seem to be enlarged. His teeth are in rather poor condition. His lower front teeth are retruded. When closing them the lower midline is to the left by the width of one lower incisor. There is a negative overjet of quite a few millimetres (Fig. 70c–e).

Radiographs. In the panoramic view (Fig. 70f) the mandible seems very strong with rather stretched but still rounded angles, the right more so than the left. Both condyles and necks are very enlarged, the right side condylar neck being longer and thicker than the left. On both sides the mandibular canal runs very close to the lower border in the first molar and bicuspid regions, on the left side more than on the longer right side. The trabecular structure is within normal limits bilaterally.

The TMJ–ramus tomograms are of rather poor quality (Fig. 70g, h). They show no real condyles. The upper ends of the thick necks are covered with strikingly thick cortical plates. There is nothing resembling an anterior “beak” and no flatness of the upper end nor are the glenoid fossae flatter than normal. On each side, the elongated necks including the condyles are in one line with the posterior border of the ramus.

The mandible in the p.a. projection at maximum opening (Fig. 70i) shows, apart from the macromandibulism with unequally long rami, very coarse trabecula-

tion within the large condyles which seem of normal shape in this particular projection.

The lateral cephalogram (Fig. 70j) shows a facial skeleton which could be called typical of acromegaly, with a rather retrodisplaced maxillary base of normal length (retromaxillism). The upper front teeth protrude and in spite of that there is a negative overjet of 11 mm. The lower front teeth are tilted moderately lingually. The large mandible has no real angle protuberances. The sella turcica seems rather small.

Scintigraphy was not requested.

Endocrinological Screening. Because of the possible existence of acromegaly, endocrinological screening was requested, in spite of the fact that such an investigation had already been done in the patient's home country, with a negative result. All laboratory values were normal and no indication was found for a possible diagnosis of acromegaly.

Provisional Diagnosis. Asymmetric bilateral hybrid form of H.H. and H.E. in a case suspected of being acromegaly.

Treatment Plan. Our usual type of planning on the tracing of the lateral cephalogram was applied and the achievable occlusion was checked on the model operation. The planning gave indication for an anterior repositioning of the maxilla by 10 mm and repositioning of the mandible by a bilateral ramus sagittal splitting procedure and advancement of the chin prominence, combined with some height reduction.

Actual Treatment. The surgery was carried out as planned, using wire fixation for the maxilla and the chin, and circumferential wiring in the rami areas, with 6 weeks intermaxillary fixation. The jaws were fixed together in a satisfactory occlusion (Fig. 70k).

Postoperative Course. Although the intermaxillary fixation was done in the planned occlusion leaving a good overjet and although there was no pressure from the tongue, 4 1/2 months after removal of the intermaxillary fixation the patient already had a negative overjet (Fig. 70l). More than 1 year later the negative overjet was the same. That means that the relapse had occurred within a very short time.

Discussion. Since we have to accept the existence of unilateral hybrid forms of H.H. and H.E., it must also be possible that such hybrid forms may develop bilaterally.

There is no question that this is a possibility in this case, in particular since the hyperactivity produced two different hemimandibles with a clearly longer right side, stretched and still roundish angles with a large radius and rather large condyles with thick necks.

Another possibility was that this case had a large mandible as the result of an anlage-induced macro-

mandibulism which was influenced in addition by a right side H.E.

The third possibility could have been that this macro-mandibulism was the result of a bilateral H.E. of asymmetric intensity in addition to a generally strong skeletal frame.

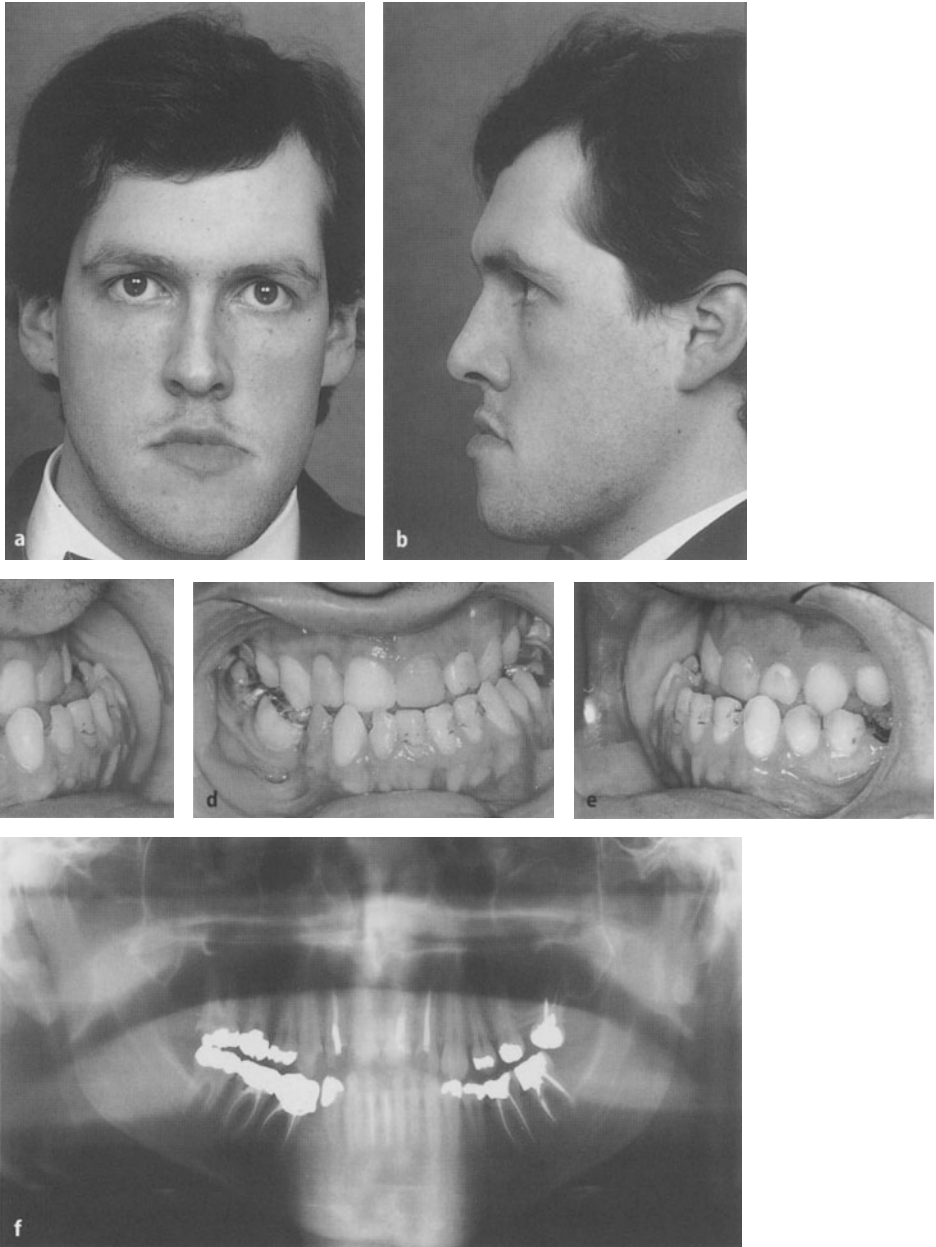


Fig. 70a-f. Bilateral, asymmetrical, questionable hybrid form with relapse after surgery. **a, b** Front and profile appearances of the patient resembling an acromegaly, but with severe chin deviation. **c-e** The patient's occlusion with deviation of the midline to the left by the width of a lower incisor. **f** In the panoramic radiograph the mandible looks strong and almost symmetrical with large condyles and thick necks, the right one a little elongated. The angles seem rather stretched

And what about the fourth possibility of a unilateral H.E. on the basis of an acromegalic enlarged mandible? The very small sella turcica seemed to speak against this. But it is known that even with a small sella turcica, an active micro-adenoma of the pituitary gland may produce acromegaly. In that case the endocrinological findings should be positive. There is also the question whether intermittent activity of such a micro-adenoma may exist as known in cases of morbus Cushing (Landolt 1999, personal communication). And if it were a case of so-called acromegaloid then the clear relapse within 4½ months could hardly be explained, in particular since the maxilla had also been moved forward by 10 mm.

I am of the opinion that relapse after surgery only occurs due to wrong planning or faulty surgery, or when the cause of the anomaly is still active at the time of surgery or is reactivated. Otherwise one can expect a stable

result. The cause of the relapse in this case is not clear. I do not think that my planning was wrong nor my surgery any worse than in other similar cases when I have achieved stable results. This means that again an unknown aetiology had caused the relapse within the space of less than half a year. It could not be the tongue as there was no macroglossia. This would tilt the teeth only but cannot bring the mandible forward. Whether or not bilateral condylar hyperactivity was still present we do not know as we had not sent the patient for scintigraphy.

Since the symmetrical forward movement of the mandible occurred within 4½ months of surgical correction where a good overbite was achieved, I do not consider that the relapse was produced by a bilateral condylar hyperactivity. If it were due to H.E. influence it would not be expected to be as absolutely symmetrical as it was.

In my mind there still remains the possibility of the existence of active acromegaly. Many years back I had

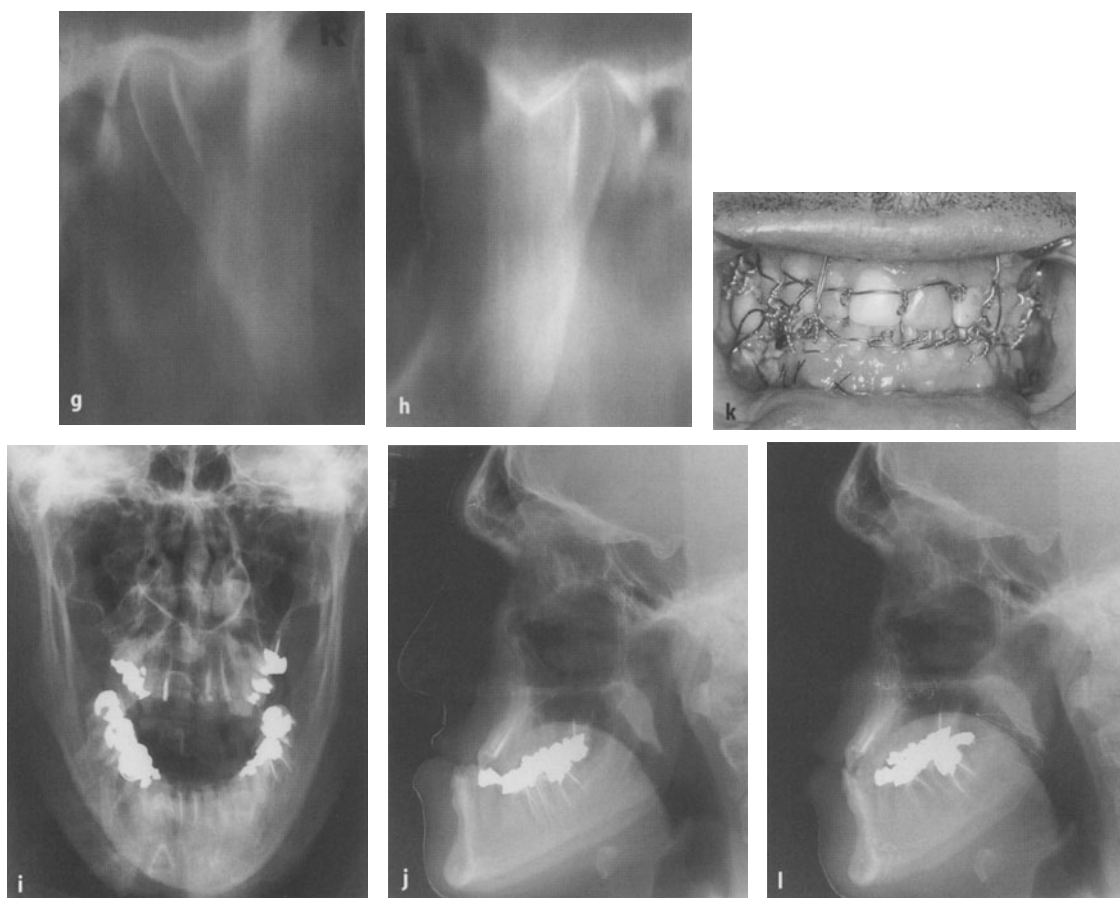


Fig. 70g–l. Bilateral, asymmetrical, questionable hybrid form with relapse after surgery. **g, h** The TMJ ramus tomograms are of rather poor quality. There are no real condyles, but equally thick long necks. **i** The mandible p.a. projection at maximum opening discloses nothing special apart from the asymmetric macromandibulism. **j** The lateral cephalogram bears a likeness to an acromegaly. But the sella turcica seems smaller than normal. **k** Fixation of the maxilla and mandible in a fairly good overjet position after bimaxillary repositioning. **l** Lateral cephalogram taken 4½ months after release of the intermaxillary fixation already shows a relapse

corrected a similar case in which I also thought that acromegaly was the cause of the antemandibulism. It was a young lady who had many more clear signs of acromegaly such as thick fingers, coarse skin, a large nose, etc. She, too, I had sent presurgically for endocrinological investigation for possible acromegaly. She too came back with a negative result. I performed the mandibular set back operation and she had complete relapse within a few months. As I had no other idea for the relapse than the possibility of still existing excessive growth hormone production, I sent her again to the department of endocrinology. Now she came back with a positive result and the suggestion of surgical treatment for her pituitary gland adenoma. Some months after her hormone values had returned to normal, I operated upon her again, using the same mandibular set-back operation – the sagittal splitting procedure on the rami followed again by 6 weeks intermaxillary fixation. No relapse occurred. Again comes the question whether or

not an already inactive acromegaly can be reactivated by an intervention or occur spontaneously. Regrettably, the patient with the non-corrected relapse disappeared to his home country.

Conclusion. This case teaches us that there is also the possibility of a bilateral asymmetric hybrid form. If the facial skeleton looks similar to acromegaly it is essential to have an endocrinological screening done, as well as scintigraphy of the facial skeleton. If there is condylar hyperactivity it would become visible and, hopefully, if there is still surplus output of growth hormone from the pituitary gland it can be proven. Even in the event of relapse and in spite of negative laboratory findings one must check them again. There must always be a reason for a relapse, according to the principle: *“If you do not remove the persisting cause of the facial skeletal anomaly, the relapse of your corrective surgery is preprogrammed”* (Principle No. 31).

14.1

Treatment Guide Lines for H.H. and Hybrid Forms

Primarily and first of all, one has to know whether the H.H. or the hybrid form is still active or whether it is burned out. Scintigraphy is the method of choice to find this out. This is valid independent of the patient's age. When the abnormal growth on the affected side has already come to a standstill, the surgery will depend on two indications, the age of the patient and the severity of the deformity.

In cases in which the abnormal growth of the mandible has come to a stop before the maxillary growth has ceased, one might wait, except in those severe cases in which the facial deformity may lead to social-psychological problems. One should not wait so long that the facial deformity can become engraved on the patient's psyche. I have often found that up to the age of 18–20

years, patients soon forget that they had suffered socially and professionally because of their facial deformity. After the age of 21–25 years, corrective surgery may produce most pleasing results and yet the patients are often unable to overcome their psychological problems completely. For that reason I am of the opinion that one should not delay the surgical treatment except only in very moderate cases. In these moderate or mild cases, correction of the already existing asymmetry can be achieved later in close cooperation with the orthodontist. Also, the very early orthodontic attempt to stop or at least influence the abnormal growth force or to bring the teeth arches into correct position does not really produce a positive result as far as further growth is concerned (see Fig. 69).

14.1.1

Treatment During the Hyperactivity Phase

When the diagnosis of H.H. or a hybrid form is clear in the growing period between childhood and puberty, one has to expect further abnormal growth of the affected side. In a very mild case one may wait and see. In the case of rather rapid growth, the main important thing is to eliminate the cause. Intracapsular high condylectomy must be carried out. Naturally, condylectomy should not be performed without prior scintigraphy (G. Paulus et al. 1987). The change in the clinical picture and the scintigraphy findings within 6–12 months can give a clear indication of the type of treatment necessary.

If high condylectomy is performed early enough, that might be all that is required, with the exception of orthodontics. Once the anomaly has produced a certain amount of difference in growth between the two sides, than condylectomy alone will not be sufficient. The condylectomy will correct a part of the asymmetry only.

Final correction of the asymmetry is best left until maxillary and mandibular growth have ceased. In cases of a very severe asymmetry in childhood, it might be wise to transplant the surplus of the inferior bowing of the mandibular body of the affected side to the contralateral side in addition to doing the high condylectomy.

The surgeon will have to identify the position of the mandibular nerve canal on the panoramic radiograph and free the nerve primarily, before cutting off the inferiorly bowed surplus of the affected mandible. It can be transplanted to the lower border of the other side or added laterally on the same side. This is all done via the oral route. However, when the permanent teeth are not fully erupted some correction of the already existing asymmetry can be better achieved by an onlay graft to the lower border of the mandible on the non-hyperactive side. I prefer to use lyophilized human cartilage for that.

14.1.2

Treatment After Growth Activity Has Ceased

After general growth and the abnormal mandibular growth have ceased, the severity and type of the deformity dictate the type of correction. It will rarely have to be performed before general growth is complete. Generally, the hyperactivity does not stop before general skeletal growth has ceased.

The vertical surplus on the affected side caused by the downward growth of the mandible and its lower border convexity and as a consequence also downward growth of the maxilla of the same side can, together with the contralateral secondary deformity of the mandibular body with its outward rotation and reduced height, produce a quite difficult surgical correction. This is particularly true when a strong hyperactivity has started very early in the patient's life. In addition comes the very awkward position of the teeth and the occlusion.

In some of the classic cases, the standard surgical treatment will be condylectomy on the affected side and transplantation of the inferiorly bowed surplus of the affected side to the contralateral side (see Figs. 31 and 37). In cases of very high vertical increase in the ascending ramus, the condylectomy alone will not produce enough reduction in its height. An additional sagittal splitting procedure of the ramus on the same side may be necessary for shortening the surplus length.

In some of the cases, when the maxilla has grown far inferiorly on the affected side, a rotation of the lower half of the facial skeleton will be necessary also. That

In a very fast growing case of H.H. or hybrid form, the maxilla will not grow inferiorly within a short time. In this situation there will be no need to move the maxilla upwards on the affected side. I personally would not perform a Le Fort I-type osteotomy for the correction of a downwardly grown maxilla as long as the permanent teeth have not completely erupted. There will be time for that later. But the patient must be clearly informed about the surgeon's philosophy and his treatment plans. Corrective surgery on the maxilla before its growth has ceased will reduce its further growth capacity.

means that the maxilla is repositioned with its occlusal plane at the planned level, parallel with the pupillary line. The mandible is moved and fixed into occlusion after bilateral sagittal splitting of the ascending rami.

In the cases in which the abnormal growth has ceased I prefer not to perform a complete condylectomy because of its consequences. There will be asymmetric mandibular movement and possible damage to the non-operated joint. Whenever possible in such cases I correct the asymmetry by osteotomies only, without touching the joint. At most I will start with a high intracapsular condylectomy.

The prominence of the chin may need additional correction if it is not automatically corrected by the transplantation of the lower border of the mandible of the affected side to that of the other side.

In non-active cases of H.H. or hybrid forms it may not be necessary to perform a condylectomy in addition to the upward rotation of the lower half of the facial skeleton. The amount of reduction in height of the ramus by the condylectomy may be compensated by the sagittal splitting procedure. However, in cases with a rather gross condyle and elongated condylar neck, both condylectomy as well as the rotation of the maxillo-mandibular complex may be necessary. This and the transposition of the mandibular lower border to the other side will be done in one surgical intervention.

14.1.3

Orthodontic Treatment

There is no point in orthodontic treatment as long as the H.H. and the hybrid forms are hyperactive. At that stage it can only make the later surgical correction of the asymmetry more difficult. Only when the final corrective surgery of the asymmetry is contemplated must the case be discussed between surgeon and orthodontist. There is no sense in planning that as long as the abnormal growth of the mandible is not burned out, either by itself or through a high condylectomy.

The main goal of presurgical orthodontics will then be to bring the teeth into proper angulation to the respective jaw base, the uppers as well as the lowers and to produce two dental arches which will fit together. As soon as the teeth are in that correct position the surgeon can perform the surgery. The whole planning of presurgical orthodontics and surgery is best done on models, by the orthodontist and the surgeon sitting together.

Bilateral Combination Forms

Apart from the possibility that bilateral forms of the same growth anomaly type exist, there is also the possibility of different mandibular growth anomalies of the two hemimandibles. Several combinations are possible: unilateral H.E. and contralateral H.H. or hybrid form. In addition there can be a combination of one of them with a hemimandibular hypoactivity resulting in hemi-

mandibular hypoplasia on the other side. These combination cases are not all that easy to diagnose when one or both of them do not develop so far that there is no dubiety about the diagnosis. Rudimentary forms are more difficult to diagnose. The following cases present some of the problems involved in diagnosis and treatment.

Unilateral Highly Active H.H. Commencing After Growth has Ceased and Contralateral Inactive Mild H.E. (Fig. 71)

Sex and Age. Female, 25 years and 10 months.

Aetiology and History. Three years previously, at the age of almost 23 years, the patient had noticed that her face was becoming asymmetrical. She had no idea why it had started, neither any recollection of previous facial trauma nor anything similar in the family history. She had consulted her dentist who referred her for clarification and evaluation and whether or not some and what kind of treatment was necessary.

Clinical Findings. Looking at the patient from in front, the difference in length between left and right sides is very obvious (Fig. 71a), the left side being remarkably longer in the vertical dimension. The lip commissure is horizontal, but the corner on the longer side is slightly more lateral than the right. The cheek area on the left side is flat without any contour. The right cheek has a normal contour with the usual slight shallow concavity between zygoma and angle region. Sensation and motor nerve activity are normal. The prominence of the chin is just out of the midline to the left. Mouth opening is normal, there is no real deviation. The difference in vertical height of the two sides of the face becomes very evident when one compares the two profile views (Fig. 71b, c).

The occlusion (Fig. 71d-f) shows a crossbite on the left side with a class II relationship on both sides. The lower incisor midline is shifted to the left by the width of slightly more than half a lower incisor. There is very little tilting of the lower incisors towards the left side.

Radiographs. *The panoramic projection in occlusion* (Fig. 71g) shows that the left mandible has a remarkably enlarged condyle with an anterior “beak”, no elongation of the condylar neck and only a moderate increase in ascending ramus height. But there is a clear vertical enlargement of the left body with a downward convexity of its lower border and a rounded angle with a very thin cortical covering. The mandibular canal runs right along the cortical plate, contrary to the situation on the right side, where the right horizontal ramus is not only less high than the left but also less than normal. The trabeculation structure is clearer in the left body area because it is stronger than on the right side. The midline of the lower teeth is shifted over to the left by almost the width of a lower incisor, when compared with the upper midline. The upper midline seems to be lying correctly in the facial midline as can be seen in relation to the nasal septum. The increase in left body height stops precisely at the midline of the chin and the midline of the lower teeth. The right lower incisors are tilted slightly towards the abnormal left side. On the right side the cheek teeth, the uppers as well as the lowers, look shorter than the ones on the other side. The right side of the mandible has a normal shaped but rather small condyle and a slightly elongated condylar neck. It shows a stretched angle and a body which seems reduced in height, particularly in the molar region. The mandibular canal runs closely underneath the molar and premolar roots, further away from the inferior mandibular

border than on the left hyperplastic side. The trabeculation is fine and unremarkable.

If one drew a line between the height of the two articular fossae, there is a vertical difference of 3 mm in the first molar region of the maxilla. This difference in height of the maxilla in addition to the difference in height of the two mandibular bodies explains the marked facial height difference.

The measurements of the tracing (Fig. 71h) of the panoramic radiograph are very interesting. The right horizontal ramus (elongation side) is 7 mm longer than that on the left H.H. side. The right ascending ramus is 10 mm shorter than the left. The difference between the length of the two trunks is even more, as they measure 52 mm on the right and 64 mm on the left side. There is also a small (1 mm) length difference between the two

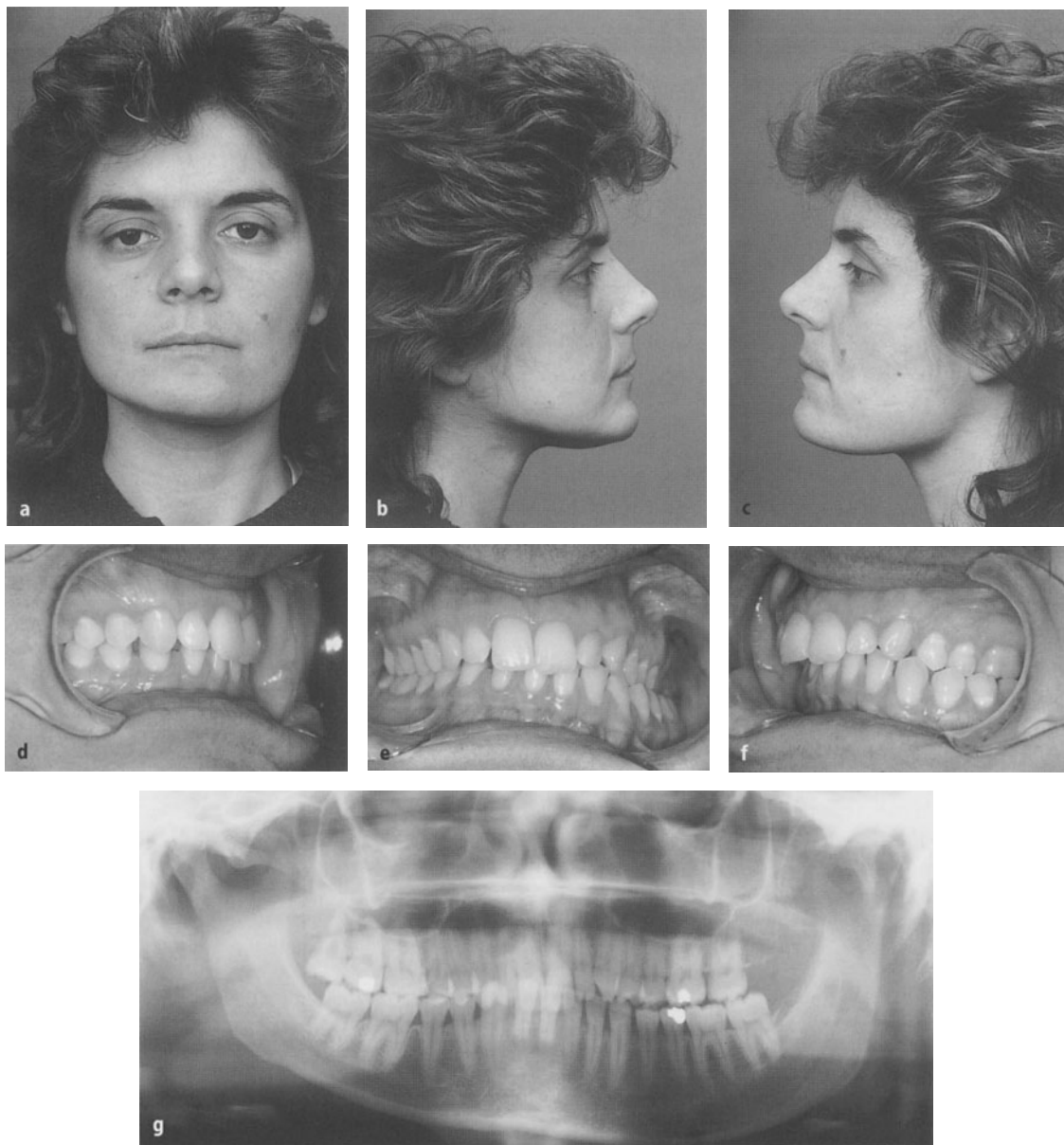


Fig. 71a–g. Unilateral highly active H.H. commencing after general growth has ceased and contralateral inactive slight H.E. **a–c** The patient's front and profile appearances showing the typical signs of a left H.H. and a right H.E. (the chin is deviated to the H.H. side). **d–f** The occlusion also permits the diagnosis of a right side H.E. and a left side H.H. **g** The panoramic radiograph permits the diagnosis of an unilateral H.H. and a contralateral H.E.

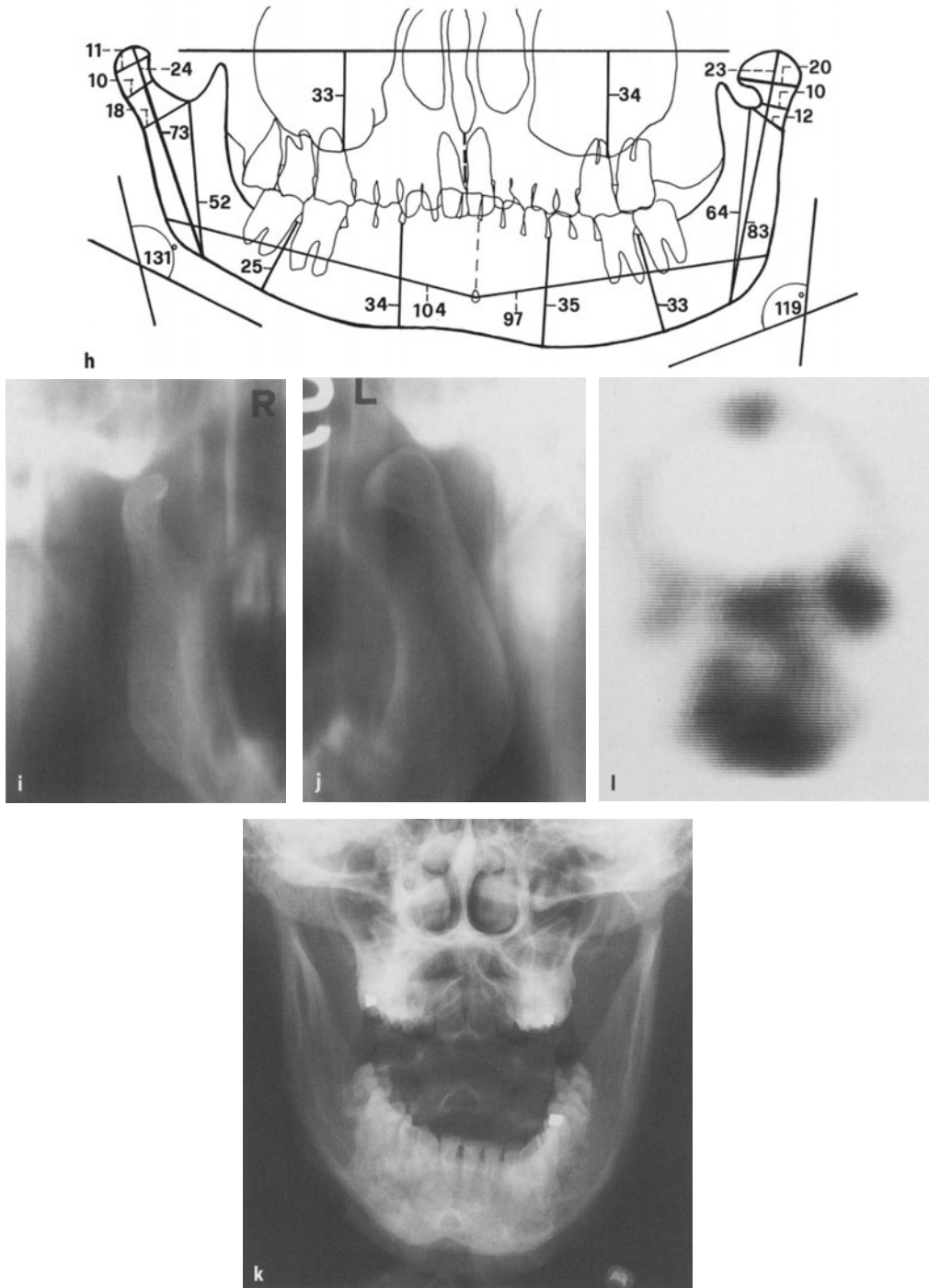


Fig. 71h-l. Unilateral highly active H.H. commencing after general growth has ceased and contralateral inactive slight H.E. **h** The measurements of the tracing of the panoramic view prove that there is a left side H.H. and a right side H.E. anomaly. **i, j** The TMJ ramus tomograms also prove the diagnosis clearly. **k** The mandible p.a. projection at maximum opening is not really significant for two different condylar growth anomalies. **l** The scintigraphy demonstrates clearly that there is very massive hyperactivity on the left side

articular processes, in favour of the H.E. side. It is rather interesting to note that the H.H. produces (in this medium degree case), more increase in ascending ramus length than does the H.E. side. In this particular case this is not due to condylar neck elongation as would be the case if it were a hybrid form. Although the tracing gives the impression of quite some difference in the maxillary height on the two sides, this is not confirmed by the measurements.

TMJ tomograms including the rami in the open position (Fig. 71i, j) show clearly the difference in shape and size between the condyles and also of the trabeculation structure of the two rami and condyles. They also show that both condyles can translate within the normal range. The left condyle is well enlarged and has the typical shape of pure H.H. anomaly. There is no elongation of its neck and no downward development of the condyle structure into the short neck. The right condyle is normal in shape and size. Its neck is rather long and slim. The gonial angle looks very stretched. The angle areas are well recognizable and measurable (134° R, 124° L).

The p.a. projection of the mandible at maximum opening (Fig. 71k) permits clear visualization of the difference in length of the ascending rami. It also shows that the mandible opens with significant deviation to the right side. There is no difference between the two sides of the maxilla.

Scintigraphy of the facial skeleton proved that there is remarkable cellular hyperactivity in the left condyle compared with the inconspicuous right side (Fig. 71l).

Diagnosis. Highly active hemimandibular hyperplasia on the left side, which had started after growth had ceased. Hemimandibular elongation on the right side of mild degree, non-nactive.

Treatment Plan. Intracapsular high condylectomy on the left side via a retrotragal approach and transplantation of the left mandibular lower border to the right side via the oral route was suggested to the patient.

Actual Treatment. The patient did not return for surgery. She was from outside Switzerland.

Discussion. In this case, the diagnosis of a left-sided H.H. was very easy, both from the patient's facial ap-

pearance as well as from the radiographs. However, there are three physical signs which do not fit the diagnosis of a pure H.H. of medium degree on the left side. One is that the midline of the lower teeth is shifted to the side of the H.H. by as much as or more than half a lower incisor, in spite of almost no tilting of the lower front teeth. The second physical sign, even more important, is the crossbite on the side of the H.H. These signs are consistent with a mild degree of right side H.E. including the shape of the right hemimandible on the panoramic X-ray view with its elongated, slim condylar neck and flat angle. The third sign is the seemingly shorter cheek teeth on the right side than those of the H.H. side, very typical of H.H. deformity.

The patient's history indicated clearly that the asymmetry did not start to be noticed by the patient before the age of 22 years. It was therefore not surprising that strong hyperactivity was still found in the condyle at the patient's present age of almost 26 years. The only proper treatment to stop further abnormal growth was high condylectomy. If the patient had come for advice when she first noticed the beginning of the asymmetry, the experienced surgeon could easily have diagnosed the beginning of a very active H.H. High intracapsular condylectomy would then have prevented the development of such an undesirable situation for the patient. In addition to the unavoidable condylectomy now the somewhat difficult reduction in the vertical height surplus of the left mandibular body is required. The consequence is that in any case of highly active H.H. high condylectomy should be performed at any age. Whether or not the slight crossbite, due to the H.E. of the other side, should be dealt with will have to be discussed with the patient. Most probably it would be corrected by a bilateral sagittal splitting procedure on the rami, and if necessary, additional orthodontic treatment.

Conclusion. H.H. can start even after general skeletal growth has ceased. As soon as a diagnosis of commencing hyperactive H.H. is made, high condylectomy must be advocated and the patient should be persuaded, if necessary with the help of photographs of fully developed cases. An already existing mild form of H.E. should not cause difficulty with the main, important diagnosis of a very active contralateral H.H.

Unilateral H.E., Slender Type, and Contralateral Hemimandibular Hypoplasia (Fig. 72)

Sex and Age. Female, 16 years of age.

Aetiology and History. This exactly 16-year-old girl had no idea what might have caused her facial asymmetry. There was nothing similar known in the family. She said the right side had always been flatter than the left. The mother reported that the chin moved out of the midline to the right side at about the age of 7 years. She did not remember the right face already being flatter in infancy. The asymmetry gradually became increasingly noticeable. At the age of 14 years she went to see an orthodontist who referred her for surgery after 2 years of unsuccessful orthodontic treatment with a monobloc appliance.

In the girl's general medical history, it must be mentioned that she had had acute tonsillitis at least 3 times a year. She did not remember either of her TMJs being painful or that she could not open her mouth properly.

Clinical Findings. The facial asymmetry is very pronounced with two main features (Fig. 72a–c). The chin is far from the facial midline to the right side, and there is a much longer left hemimandible with a rather short right one. The second striking feature is the flatness of the right side of the face. This includes the cheek as well as the zygoma-infraorbital rim region. The lip commissure is oblique, slightly ascending to the corner of the flatter right side of the face. In profile views there is not much of an abnormality to be noticed.

Mouth opening was unimpeded but with a certain amount of chin deviation to the flat right side, although much less so than when the mouth was closed. There is a clear crossbite on the right side and the lower teeth midline is shifted to the right by the width of 1 1/2 lower incisors (Fig. 72d–f). On the longer left side there is a severe class III occlusion and on the shorter right side

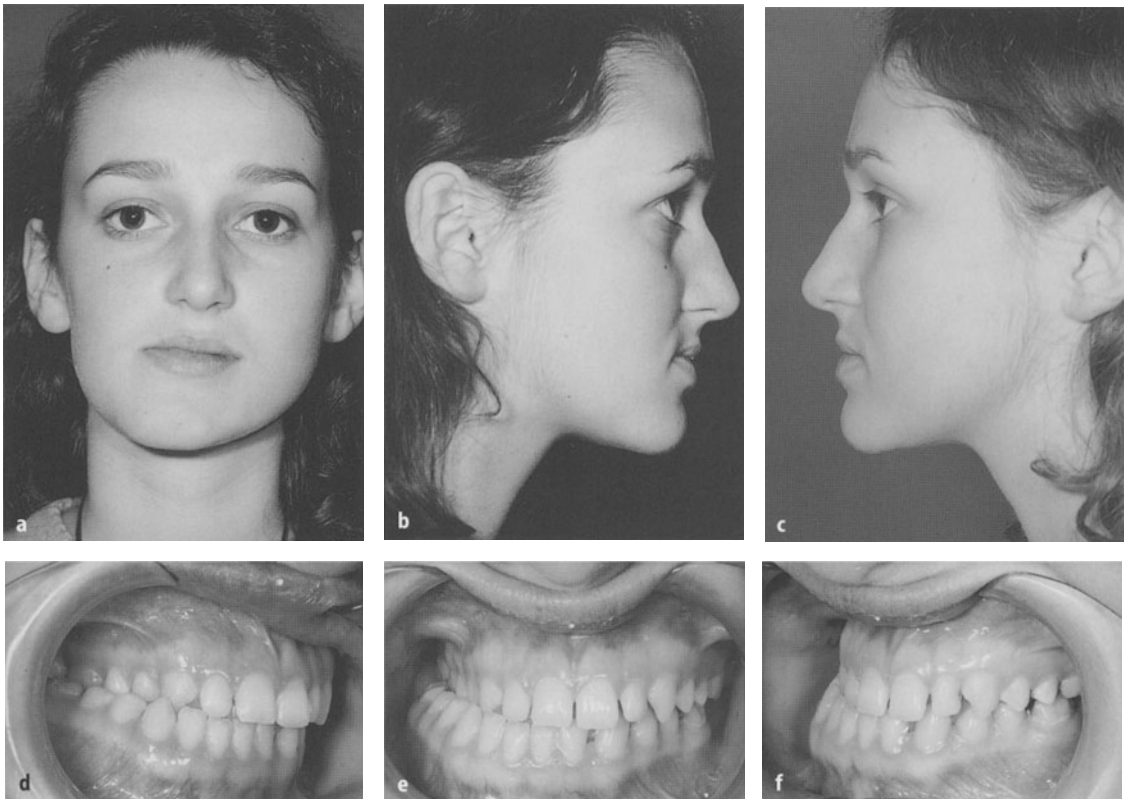


Fig. 72a–f. Unilateral H.E., slender type, and contralateral hemimandibular hypoplasia. **a–c** The patient's front and profile appearance before surgery. **d–f** The patient's occlusion before surgery with a crossbite and deviation of the midline of the lower teeth by the width of 1 1/2 inferior incisors

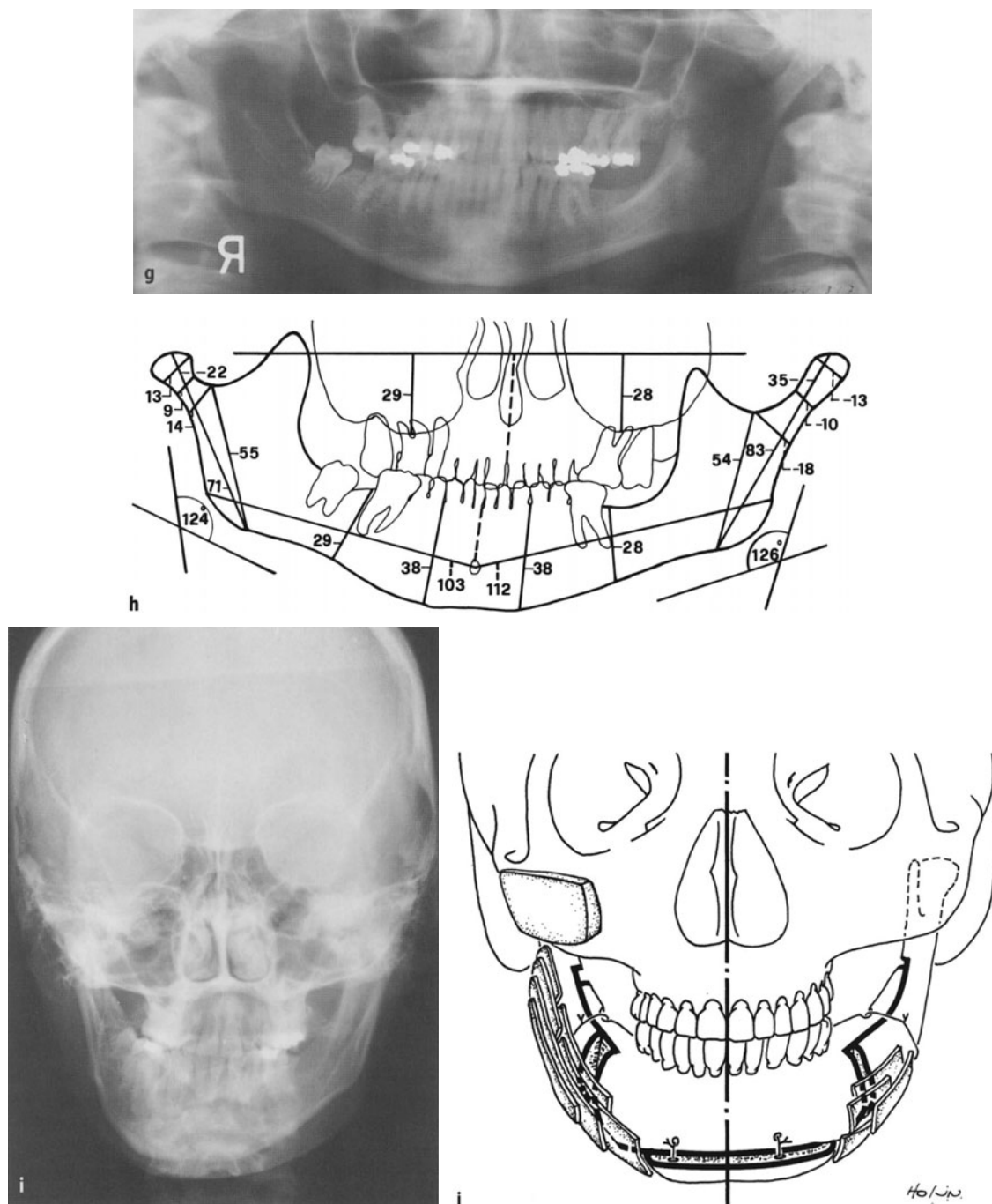


Fig. 72g-j. Unilateral H.E., slender type, and contralateral hemimandibular hypoplasia. **a-c** The patient's front and profile appearance before surgery. **d-f** The patient's occlusion before surgery with a crossbite and deviation of the midline of the lower teeth by the width of $1\frac{1}{2}$ inferior incisors. **g** The panoramic view discloses a typical H.E., slender type, on the left side and also a typical hemimandibular hypoplasia on the right side with a small sclerosed condyle. **h** The measurements of the panoramic radiograph clearly indicate why there is such severe asymmetry. **i** The p.a. cephalogram shows the asymmetry of the mandible and maxilla better than does the OPT. **j** Diagrammatic illustration of the planned correction in two interventions

there is a clear class II occlusion. There is also a well noticeable oblique occlusal plane with a vertical height difference in the first molar region of 6 mm, as measured on the orthopantomogram. Facial musculature and nerve function were normal.

Radiographs. *The panoramic view* (Fig. 72g) discloses the skeletal background of the first main feature of this rather severe facial asymmetry. There is a clearly elongated left hemimandible and a rather short right one. The condyle and its neck on the longer left side have almost the same thickness in this projection and the neck is clearly elongated. The angle on that side is stretched while the angle on the shorter side seems to be within normal limits. Both sides have a very flat antegonial depression within the range of normal, the right being a little deeper than the left. The right condyle does not have a normal shape. It appears round and small and sits on a short, rather thin neck. Its bone structure clearly shows it to be sclerosed.

The measurement results from the tracing (Fig. 72h) are not as impressive as expected: the left horizontal ramus is 9 mm longer than the right and the ascending ramus 12 mm longer which is due only to the difference in length of the two articular processes (13 mm). The height of the two trunks is about the same and also the angles do not differ by much.

The TMJ tomograms were of no use because of their poor quality.

The lateral cephalogram showed a very reasonable intermaxillary relationship.

But the *p.a. cephalogram* (Fig. 72i) demonstrates even better the mandibular asymmetry and the oblique occlusal plane, as did the OPT. It also discloses a clear rotation of the maxilla, on the left side being further inferiorly than on the hypoplastic right side.

Provisional Diagnosis. Unilateral H.E., slender type and contralateral hemimandibular hypoplasia, probably the result of arthritis.

Treatment Plan. At least two operations would be necessary (Fig. 72j): first, bilateral sagittal splitting of the rami and mandibular repositioning would be necessary to restore normal occlusion which should present no difficulties according to the model operation. A rotation of the lower half of the facial skeleton was found not to be necessary as the vertical discrepancy between the left and the right sides of the maxilla was not so severe.

The girl wanted the correction performed as soon as possible. As she was only 16 years old, there might still have been some on-going condylar hyperactivity on the left side which could have led to a certain amount of relapse. Therefore, one would either have had to wait at least a full year or more before the final correction could be done, or one could do it now with the risk of some

degree of relapse. Scintigraphic evaluation could have helped in this decision. The correction of a relapse would either have to be a bilateral rami splitting procedure again, plus repositioning of the maxilla, plus chin symmetrization as well as onlays of strips of lyophilized cartilage to the hypoplastic right body, ascending ramus and zygoma area or chin correction and placement of lyophilized cartilage strips to the right hemimandible and zygoma without a further sagittal splitting and maxillary repositioning procedure.

Actual Treatment. By a bilateral sagittal splitting procedure on the rami, good occlusion was achieved, but quite an asymmetry of the face and the chin still remained (Fig. 72k, l). Sixteen months later the chin asymmetry was corrected by a sliding osteotomy and the right-sided hypoplasia was corrected by on-laying many slices of lyophilized human cartilage on the body and the ascending ramus and the zygoma and its arch on the right side, and also some on the left body. The facial asymmetry was now completely corrected (Fig. 72m).

Twenty-four years after the second intervention (Fig. 72n-t) the face was still symmetrical, there was no deviation of the mandible on mouth opening and there was no oblique occlusal plane. The occlusion was still the same as were the facial contours in the horizontal and ascending ramus areas. That proves again that contour correction by subperiosteal implantation of thin slices of lyophilized human cadaver cartilage is a worthwhile procedure.

Discussion. I had never seen such a severe mandibular asymmetry due to an unilateral condylar misregulation of growth in a H.E. alone. It therefore automatically drew attention to a possible additional aetiology on the contralateral side. In spite of then not having the present-day easily available additional diagnostic assistance of good CT ramus tomograms and scintigraphy and even more sophisticated examinations, the bilateral origin of this severe asymmetry could be easily diagnosed with old-fashioned standard radiographs. Although the girl was informed that due to her age a relapse might occur, she wanted the operation immediately, for psychic reasons, taking into account that the same operation may have to be repeated again in 2 or 3 years when her mandibular growth, including the condylar L-growth regulator hyperactivity, had ceased. In this case no further growth occurred. The mandibular rotation, by the bilateral rami splitting procedure, produced such an improvement that an additional Le Fort I osteotomy for rotating the whole lower half of her facial skeleton did not seem to be indicated.

The lyophilized human cartilage, rehydrated with an antibiotic solution and cut into thin slices with a dermatome, again proved to be the most valuable and shape-retaining material for contour correction.

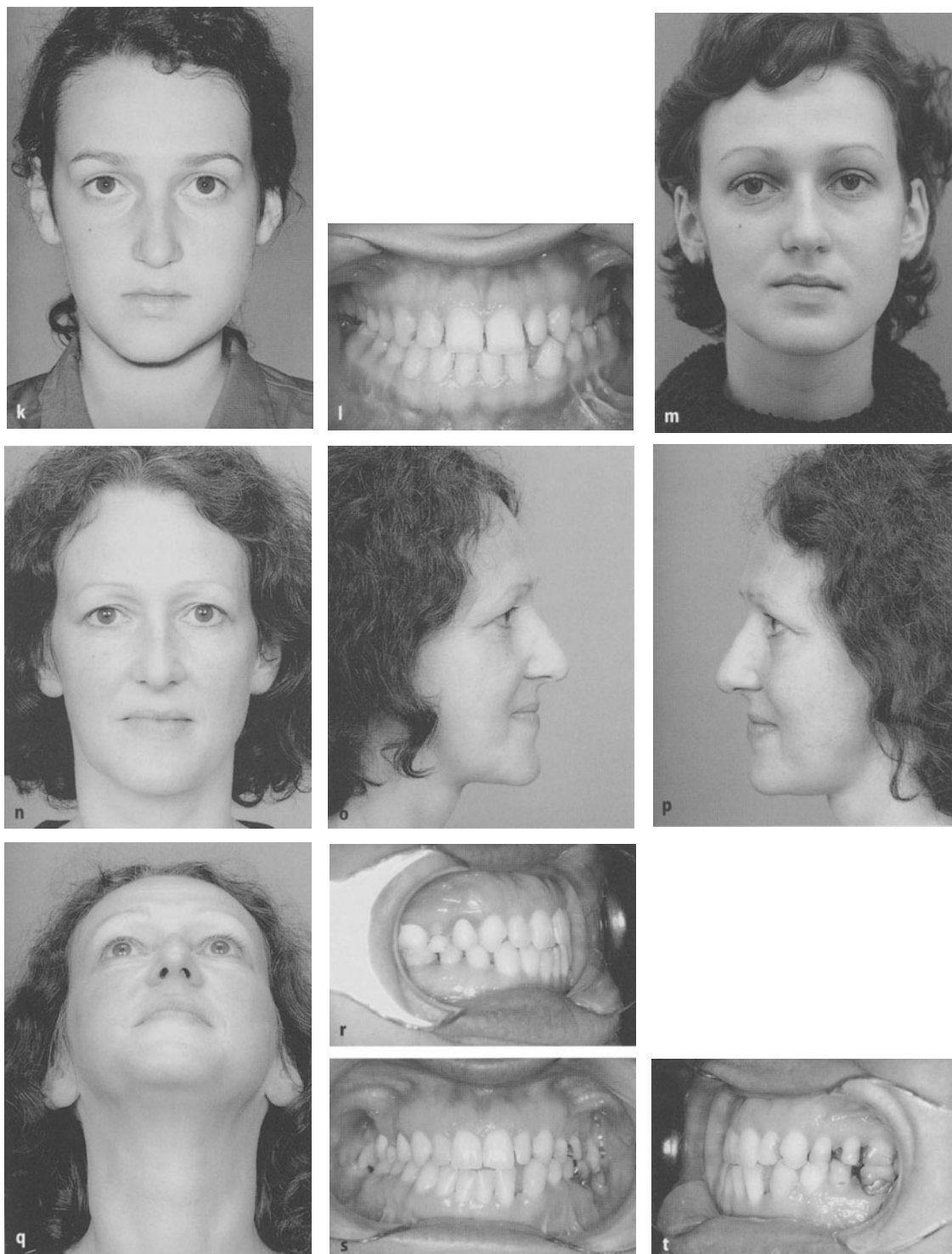


Fig. 72k–t. Unilateral H.E., slender type, and contralateral hemimandibular hypoplasia. **k, l** After bilateral sagittal splitting of the rami and repositioning a good occlusion was achieved, but some considerable facial asymmetry remained. **m** Front appearance of the patient 3 months after the second intervention. **n–t** The patient's appearance and occlusion 24 years after the second operation proves the long term stability of the skeletal correction as well as of the use of lyo-cartilage slices for contour correction

The question regarding the origin of the hemimandibular hypoplasia on the right side still remained in this case. The possibility of a congenital hypoplasia of the condyle could be eliminated, for several reasons. The sclerosis of the condyle did not fit that origin. The lack of maxillary down-growth would have been much greater if it were due to a congenital lack of growth on that side of the mandible and the antegonial notch would have been pronounced. That meant that it was due to postnatally acquired damage to the condyle at an age when the maxillary downward growth was already almost finished. The girl's recurrent acute tonsillitis throughout her childhood could easily have led to an undiagnosed TMJ arthritis, thus damaging the region of the condylar growth regulators, as has been rather often observed.

Unilateral Highly Active Hybrid Form with Gross H.E. Predominance and Contralateral Hemimandibular Hypoplasia (Fig. 73)

Sex and Age. Female, 11 years and 8 months.

Aetiology and History. The mother had noticed only a year previously, at the age of over 10, that her daughter's chin had grown out of the midline. She consulted an orthodontist who referred the girl for evaluation and treatment planning. The mother had no idea of any possible cause. There was nothing relevant in the family history. The girl had never complained about TMJ problems.

Clinical Findings. The girl is rather small for her age but in absolutely perfect health. She has a slight facial asymmetry, with the right side a little flatter than the left (Fig. 73a). This is true for the zygoma-infraorbital area as well as for the right cheek. The left side of the face seems normally contoured. The chin prominence is shifted to the right by about 8 mm. The lip commissure ascends slightly to the shorter right side. There is less vertical distance between the right eye and the right corner of the mouth than on the left side. Mouth opening is not reduced, but it deviates to the right side (Fig. 73b). There is an oblique occlusal plane, ascending to the right side (Fig. 73c). The girl has a pleasing profile line of the vertical profile type, with a bit of a retromandibulism (Fig. 73d). The facial musculature and nerves are unremarkable, except that the right masseter muscle is less voluminous than the left.

There is a mixed dentition. So far, only the two first upper and the four lower incisors and the first molars of the permanent dentition have fully erupted. The lower midline is shifted to the right by 3 mm (Fig. 73e).

Conclusion. Any mandibular asymmetry may be caused either by an unilateral surplus or a deficit of growth or it may have originated on both sides. That bilateral origin can either be an asymmetric surplus or a lack of growth or it may be an unilateral surplus and a contralateral deficit, as in this case. The early correction of such a facial asymmetry is essential if the patient begins to suffer psychically, even if a relapse may occur subsequently. Whether or not in such a case a high condylectomy should have been performed as a preliminary measure is a matter of choice. I prefer not to operate on the joint if it is not necessary. A H.E. mandibular asymmetry is quite different from that point of view to a H.H. or, even worse, a hybrid form.

Six months later, at age 11 years and 8 months, the asymmetry had increased slightly as seen from in front, as well as in the occlusion. Skeletal scintigraphy revealed clear hyperactivity on the left side compared with the right. High condylectomy was suggested, but refused by the parents.

One and a half years later, at the age of 13 years and 9 months, the asymmetry has not changed the appearance very much (Fig. 73f), but the occlusion has changed quite a bit (Fig. 73g). The lower jaw has shifted further to the right side and the occlusal plane has become more oblique.

Radiographs. The panoramic view, taken at the girl's age of 11 years and 2 months (Fig. 73h), shows the mandibular asymmetry clearly. The right ramus-condyle length is remarkably less than on the left side, but nearly within the normal range in accordance with the girl's general skeletal development, except that the condyle is rather small with a short neck and a small anterior "beak". The antegonial concavity is a little more pronounced on the right than on the left side. The left ascending ramus, condyle and neck region seem unusually strong for this girl's skeletal developmental age. Particularly striking is the thick and bulging condylar neck. The condyle itself is within the normal range. The left horizontal ramus is remarkably longer than the right, the difference measuring 14 mm on this panoramic view. The left ascending ramus measures 77 mm compared with 62 mm on the right side. The body on the right side is less high in the

molar region than on the left side. The inferior rim of the left horizontal ramus shows a slight downward convexity in the bicuspid region as compared with the right side, although not very remarkable. The maxilla on the right side is clearly less high (long) than on the left.

The panoramic view taken at age 13 years and 9 months (Fig. 73i) discloses a clear accentuation of the previously observed difference between left and right halves of the masticatory skeleton. The left horizontal and ascending rami seem to have increased in their length and mass. The left horizontal ramus now has a clear downward convexity in the premolar and molar area due to increase in bone mass between the roots of these teeth and the lower border. The left condyle has slightly increased in size and the right condyle seems to

have become smaller and its neck narrower than 18 months earlier.

The maxilla on the left side has grown further inferiorly compared with 18 months earlier.

The mandible p.a. projection in the open mouth position taken at age 11 years and 8 months (Fig. 73j) shows a clear difference between right and left sides. The right side is shorter than normal in the ramus area and the left has a long ascending ramus, in particular a longer neck. But the whole left side, from the condyle to the symphysis, is very much longer than the right hemimandible and the chin extends far over to the right, shorter side. Both ascending rami look rather straight.

The mandible p.a. projection 2 years later (Fig. 73k) in the open mouth position, at 13 years and 8 months, re-

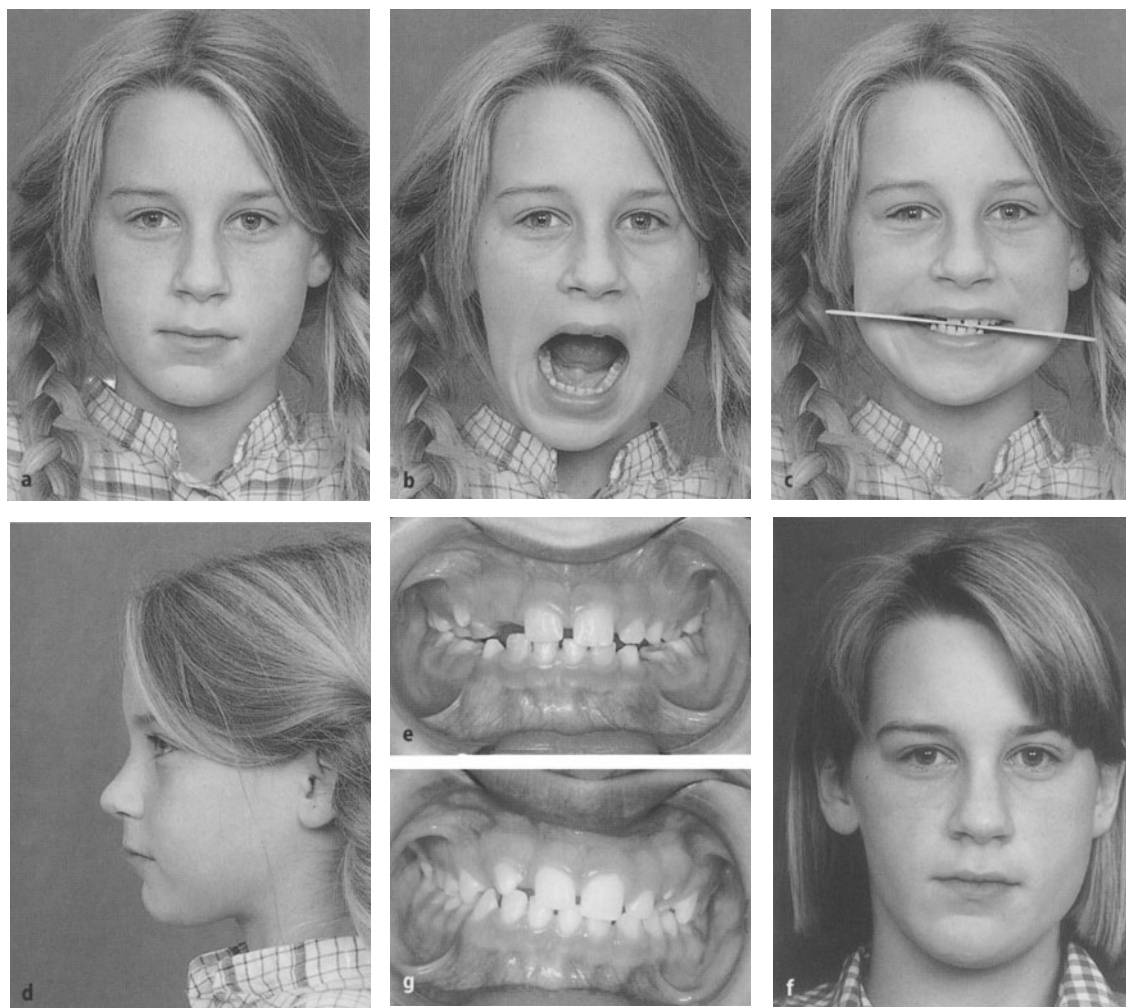


Fig. 73a–g. Unilateral active hybrid form with H.E. predominance and contralateral hemimandibular hypoplasia. **a–d** The patient's appearance at age 11 years and 8 months with asymmetry of the mandible in the closed and open mouth positions, with an oblique occlusal plane and a normal profile. **e** In occlusion, there is deviation of the lower teeth midline to the right side by half a lower incisor. **f, g** The patient's frontal appearance 2 years later shows little increase in the mandibular asymmetry, but it is clearly noticeable in the occlusion

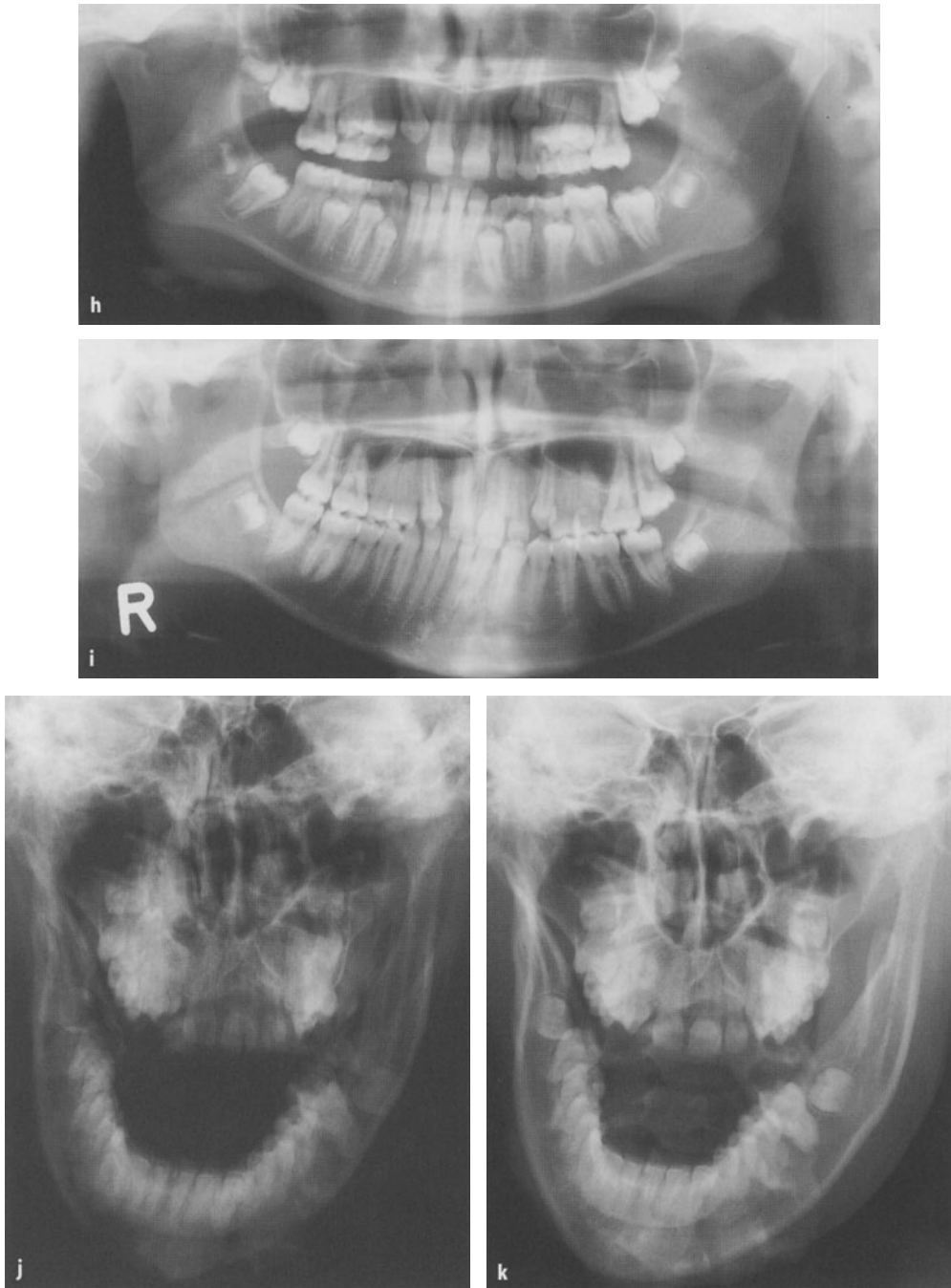


Fig. 73h–k. Unilateral active hybrid form with H.E. predominance and contralateral hemimandibular hypoplasia. **h** The panoramic radiograph, taken at 11 years and 8 months, showing a long left side with a longer and thicker neck than normal and a rather small condyle and a short right ascending ramus with a short neck and a slightly deformed condyle. **i** The panoramic view, taken 2 years later (age 13 years and 8 months) reveals a further increase in the difference between the two hemimandibles. **j, k** The p.a. projection of the mandible at maximum opening, taken at 11 years and 8 months, shows a much longer left mandible than the right and the same projection, taken 2 years later, leaves no doubt that there is further rapid overgrowth of the left side

veals some change compared with the same projection 2 years earlier. The discrepancy in length between the two ascending rami has increased. Also their shape has changed, the left is curved a bit laterally, while the right has a medial bend above the angle region. The deviation of the chin has also increased noticeably and so has the vertical height of the left maxilla, while the right side lack of downward growth is an unerring sign of lack of mandibular ascending ramus lengthening.

TMJ-ramus tomograms (Fig. 73l, m) at age 13 years and 8 months reveal a very hypoplastic right condyle with an anterior sclerotic excrescence sitting on a narrow and rather short and also partially sclerotic neck. The radiograph of the left side discloses a long articular process with a rather thick neck and a peculiarly shaped condyle of medium size without a cortical covering and with a coarse and translucent trabecular structure (as typical for the H.H. condyle) extending downwards into the condylar neck – an appearance very much like the hybrid form type.

The measurements of the tracing of that panoramic radiograph just before surgery (Fig. 73v) show that the actual differences between left and right hemimandibles are greater than one would expect clinically. The horizontal rami differ by 24 mm in length and the ascending rami by 16 mm. The height difference in the horizontal rami is also clearly noticeable and even more so between the trunks (9 mm). The same is true for the condylar processes (6 mm) and the height difference between the two sides of the maxilla, measured at the AL-points in front of the first molars (4 mm).

The Institute for Nuclear Medicine and Radiotherapy (Head: Prof. Dr. W. Horst) did full skeletal *scintigraphy* with 500 MBq Tc-99m-DPD. A 20% higher cellular activity was found on the left side compared with the right (Fig. 73n).

Provisional Diagnosis. Active hybrid form on the left side with H.E. predominance and hemimandibular hypoplasia on the right side.

Treatment Plan. At age 11 years and 8 months, high intracapsular condylectomy on the left and sliding transoral chin correction were proposed, with postoperative use of cast cap splints with training flanges for 3 months if they stayed firmly in place in spite of the teeth being small. The final correction of the facial asymmetry to be performed when the girl's general growth had ceased. A rotation of the lower half of the facial skeleton would become necessary with additional contour correction. The patient had to remain under orthodontic observation until the final surgical correction.

The parents refused to have the operation done and wanted to wait, but agreed when they saw that the asymmetry had increased quite remarkably within 2 years.

Actual Treatment and Postoperative Course. Intracapsular high condylectomy was performed at the age of 13 years and 10 months via a retrotragal approach. The planned sliding genioplasty correction was done also.

Cast cap splints were cemented postoperatively, their training flange causing the girl to close into perfect occlusion without deviation. The splints stayed firm for 6 weeks.

A *TMJ tomogram* taken 1 week after the high condylectomy shows clearly the line of resection on top of the condylar neck (Fig. 73o): 2 years and 8 months later the condyle has regenerated to an almost normal shape, but smaller in size (Fig. 73p). As a consequence of the high condylectomy and sliding chin correction, and probably in addition due to the cast cap splints with a training flange, a very satisfactory early result was achieved. There was not yet, at the age of 13 years and 10 months, complete correction of the asymmetry.

Five months after the intervention there is still some flatness of the right facial side and some chin deviation (Fig. 73q). There is some deviation on maximum opening, but much less than before the high condylectomy and not to the side of the high condylectomy, as one might have expected, but to the right hemimandibular hypoplasia side (Fig. 73r). The girl can move her lower jaw to both sides almost equally and forwards without deviation (Fig. 73s, t).

A *panoramic radiograph* taken 2 years and 8 months after condylectomy (Fig. 73u) discloses that the mandible has undergone quite some change.

The comparison of the measurements of the tracing of the OPT taken just before condylectomy (Fig. 73v) with the new one taken 2 years and 8 months after condylectomy (Fig. 73w) proves it, although the surgery itself has not changed the horizontal or ascending rami, except by the amount of the high condylectomy.

The two horizontal rami are now almost equal in length. Before surgery there was a difference of 24 mm and now only 7 mm, in spite of the fact that the later panoramic view measured on the tracing 10 mm longer from G-point to G-point than did the presurgical one. Also the difference in length of the ascending rami has changed from 16 mm to a difference of only 6 mm. A new left condyle has regenerated to an almost normal shape, smaller in size than normal, but almost equal to that of the hypoplastic right condyle. That means that altogether the two hemimandibles had undergone so much spontaneous change that they had come closer to being alike.

Histology. Regrettably, only photomicrographs taken earlier were available for histological examination. These showed that the articular surface of the removed condyle, wherever it was preserved during surgery, was formed by a rather thick articular layer. Whereas the subjacent proliferative layer was difficult to recognize, the deepest layer of hypertrophic growth cartilage was

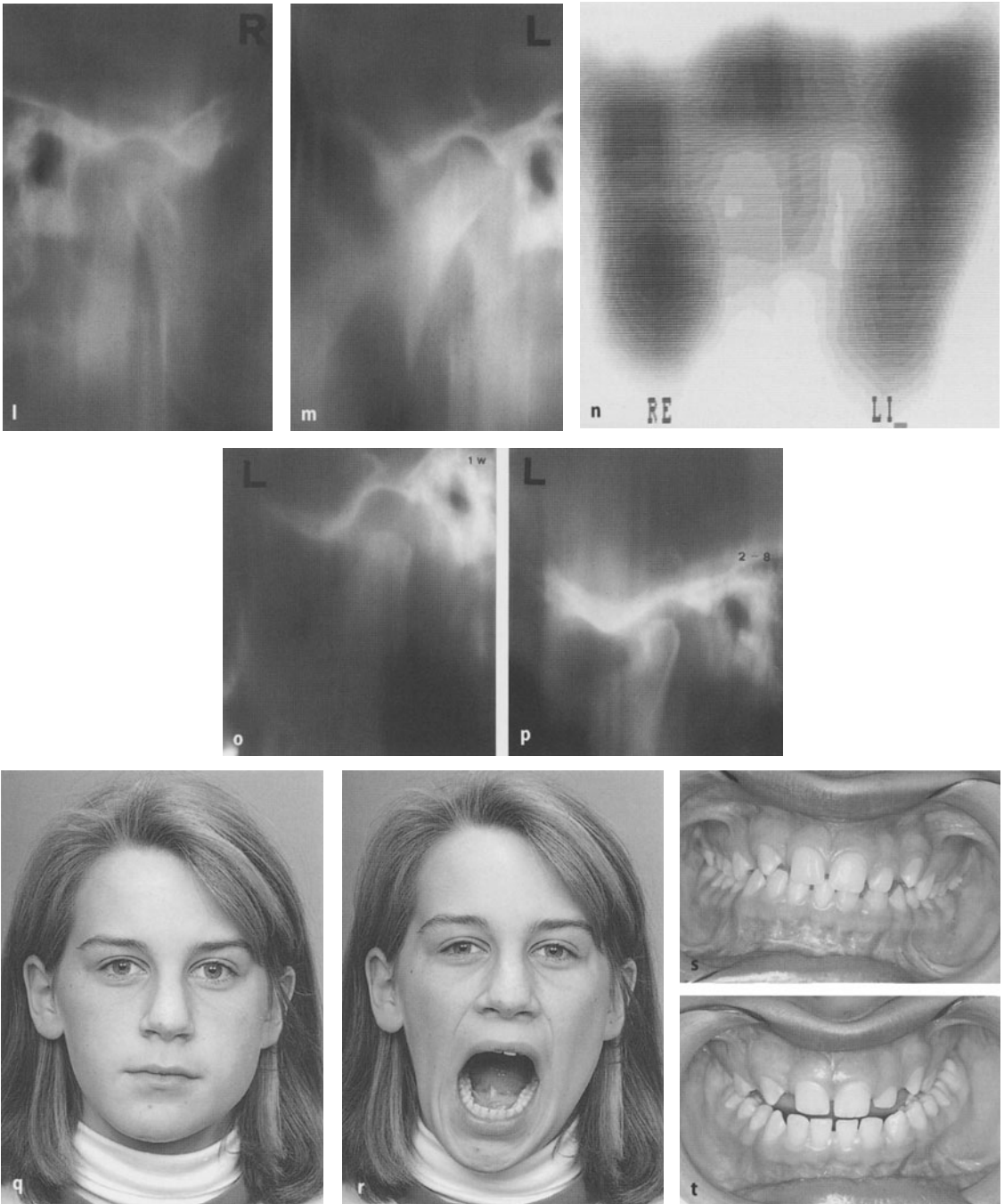


Fig. 731-t. Unilateral active hybrid form with H.E. predominance and contralateral hemimandibular hypoplasia. **l, m** The TMJ ramus tomograms, taken at age 13 years and 9 months, confirm that both condyles are not normal in size and shape. There is irregular hypoplasia on the right side and long and thick neck, and a large and deformed condyle on the left side. **n** The scintigraphy confirms clear hyperactivity in the left condyle. **o, p** TMJ tomograms from the left side, the one taken 1 week and the other 2 years and 8 months after condylectomy: a small new condyle has formed spontaneously. **q, r** Eleven months after high condylectomy only and chin repositioning, there is still some facial asymmetry. Mouth opening is within the normal range, with less deviation than before. **s, t** Occlusion and unimpeded forward movement without deviation 1 year and 2 months after condylectomy

again prominent. Endochondral ossification was apparently highly active, as almost the entire lower border of the hypertrophic cartilage was in contact with marrow spaces. The subchondral spongiosa strikingly resembled that of the other H.E. cases (see Fig. 79c, e). Relatively large marrow spaces were arranged between few, slender, almost parallel bone trabeculae containing cartilage islands.

Diagnosis. Actively growing condyle exhibiting a conspicuous spongiosa characterized by few slender, almost parallel trabeculae typical of H.E.

Discussion. According to our patient population, we do not doubt that in any hybrid form of H.H. and H.E., all gradations of hyperactivity of the M- or L-growth regulator may develop. In a case in which the elongation

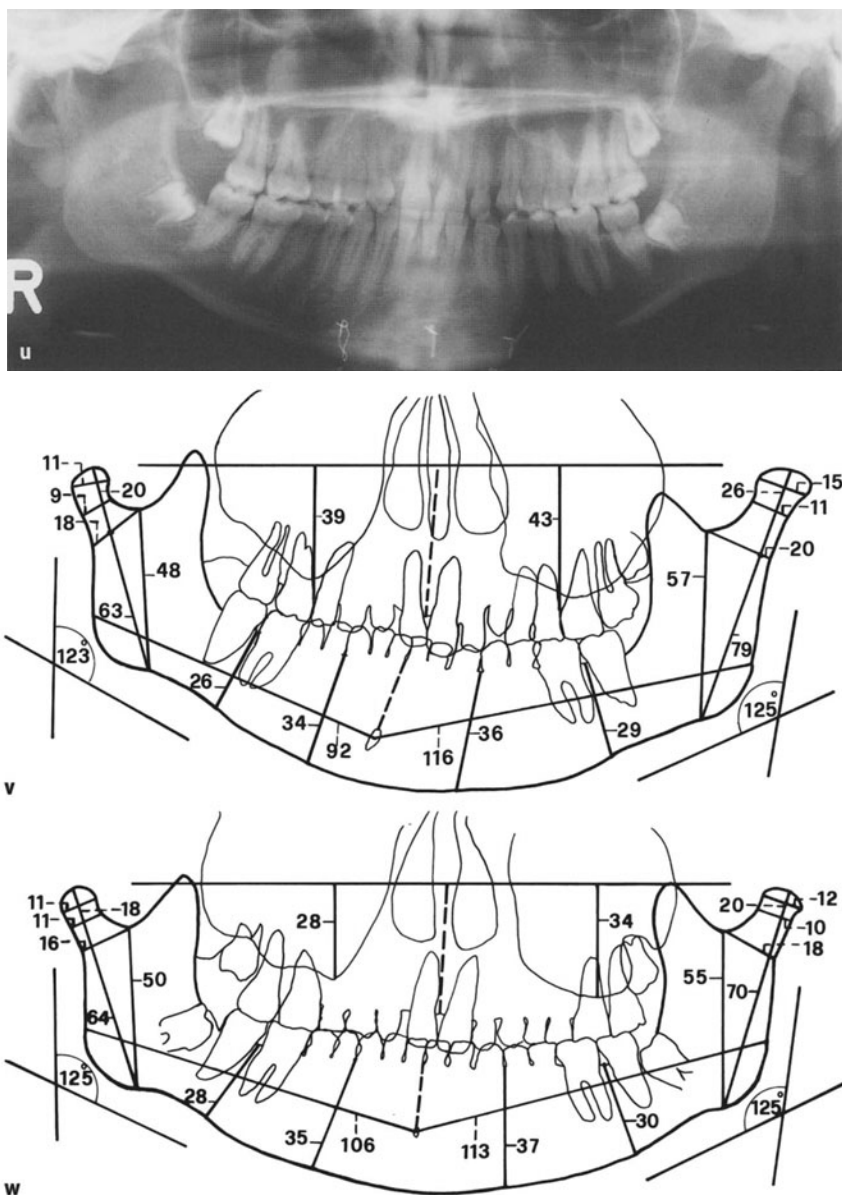


Fig. 73u-w. Unilateral active hybrid form with H.E. predominance and contralateral hemimandibular hypoplasia. **u** Panoramic view, taken 2 years and 8 months after surgery, discloses that the two hemimandibles have changed spontaneously so much that they now look almost equal. **v** The tracing of the panoramic radiograph, taken a day before surgery (Fig. 73i) discloses rather larger differences between the left and right hemimandibles than one might have expected. **w** The tracing of the panoramic view, taken 2 years and 8 months after surgery (Fig. 73n), discloses that such an amount of change had taken place that both hemimandibles look now very much alike

seems to be predominant, it might easily happen that a contralateral hypoactivity is overlooked. Once the development has proceeded so far that one can definitely diagnose a lack of downward growth of the maxilla on the questionably hypoplastic side, therein lies the proof.

There is no question that high condylectomy is the correct early treatment for any hybrid form, independent of which of the two growth regulators is predominant. But a case with a hemimandibular hypoplasia that prevents the maxilla growing down will finally always require a rotation of the lower half of the facial skeleton in order to achieve a good functional and aesthetic result. Practically all cases in which a rotation of the lower half of the face is indicated, correction of the chin asymmetry is also an important part of the rotation of the lower half of the facial skeleton. It might be a question whether or not one should do a Le Fort I osteotomy for facial rotation at the age of 13 years. As long as no permanent upper teeth will be damaged, one might do it at that age and willingly accept a possible later secondary correction because of possible further growth of the mandible. This is definitely better than having young girls suffering psychologically because of their facial asymmetry.

It is interesting to note that spontaneous regeneration of the condyle had taken place within 2 years and 8 months, not as the same hyperplastic type but as a roundish rather small condyle. Whether this was due to the postoperative functional treatment with the cast cap splints or just by chance is difficult to say. It definitely did not happen in all our cases after high condylectomy. I am convinced that these splints assist the mandible remarkably in its movements, ending with a perfect occlusion as well as no asymmetry in forward movement. Also, the maximum mouth opening showed only a little deviation to the hypoplastic right side and not to the side of the condylectomy as one might have expected.

Measuring the tracings of the panoramic views can elucidate very interesting points. It not only confirms the differences between left and right sides but it shows that in this case quite an amount of spontaneous change in the shape of the two hemimandibles has taken place: The length of the right ascending ramus and the height of both horizontal rami have remained practically unchanged. But not so the length of the horizontal rami. The right has increased in length by 14 mm and the left by 3 mm. The left ascending ramus has decreased in length by 10 mm although the actual amount lost through the high condylectomy may be 4–5 mm at most. The explanation for these facts cannot only be that two panoramic views are not comparable, because the relationship between the length of the left and right horizontal rami has changed. The horizontal ramus of the condyle resection side has lost 3 mm in length while that of the hemimandibular hypoplasia side has gained 14 mm. The only explanation for this may be that the hypoplastic side has gained length in its horizontal ramus through physiological growth. But in the horizontal ramus only, while its trunk gained 2 mm only? The change in the other hemimandible, the reduction in length in its horizontal ramus by 3 mm and of its trunk by 2 mm may be a result of remodelling due to the functional matrix after the push from the condylar growth factors had been eliminated. I do not know the answer. It is just an attempt to find an explanation for the changes which obviously have happened.

Conclusion. The combination of a hyperactivity anomaly on each side can cause diagnostic problems when they are rather similar in their signs. Exact measurements of tracings of the panoramic radiograph may help in understanding the anatomical facts.

This case proved that there may be a spontaneous regeneration of something like a new condyle. This does not happen in every case of high condylectomy.

Unilateral Questionable Hybrid Form on the Left Side and Contralateral H.E., Slender Type (Fig. 74)

Sex and Age. Female, 40 years of age.

Aetiology and History. The patient could not recall any possible cause for the asymmetric growth of her mandible. There was nothing similar known in the family history. It first became noticeable at the age of 14 and then developed progressively until the age of 20. From then on it stopped, but maybe not completely, she said. Around the age of 30 she experienced severe pain in the whole right side of the face and neck, with pain-free intervals that became shorter and shorter. The pain and the aesthetic-psychic problems induced her to seek correction.

Clinical Findings. In the front view, a rather severe facial asymmetry is noticeable, with the left side remarkably longer in the vertical dimension than the right (Fig. 74a–d). The left side is flat with the lower border of the mandible much lower than the right. It is slightly downwardly curved and directed medially while the right side is shorter in the vertical dimension and tilted slightly laterally. The symphysis of the chin is almost in the facial midline and the left side of the chin is more prominent than the right. The patient's facial asymmetry is particularly striking when looking at her face from below. In profile view the differences in vertical dimension become very obvious. The facial soft tissues and innervation were undisturbed. Mouth opening is not restricted, but deviates on opening to the right side.

The midline of the lower teeth is 3–4 mm over to the left of the upper midline. The right molars are in a class III occlusion and on the left side there is a slight open bite all the way back from the canine region. In the molar region of the enlarged side there is a class II and a slight lateral crossbite situation. On the right side the teeth seem shorter due to lingual tilting than those on the left side which remain in a vertical position. In addition, the lower front teeth are tilted to the left. The whole occlusion appears twisted (Fig. 74e–g).

Radiographs. The panoramic view in the closed position (Fig. 74h) discloses the discrepancy between the left and right sides very clearly, with typical enlargement of all sections of the left mandible. The condyle is quite enlarged but not with an anterior “beak”. Its neck seems elongated but not thicker than normal, its ascending ramus including condyle is much longer than that on the other side. Its posterior border is vertical, its angle rounded, its body higher than normal and its lower border rather convex downwards. There is coarse trabeculation in the left body and ascending ramus regions, while the condyle shows areas of sclerosis and

translucency. The mandibular canal is far down, close to the lower border which seems to have a thinner cortical plate than that on the other side. The right side is rather slim with a stretched angle and lack of body height in the molar region and a rather small condyle with a slightly elongated neck. The symphysis is noticeably, but not much, out of the facial midline to the left side.

The measurements of the tracing of the panoramic view (Fig. 74i) indicates that the horizontal ramus of the left H.H. side is 11 mm longer than that of the right H.E. side. As the Pg-point is almost exactly at the midline of the face, it suggests that the difference must be due to the different orientations of the posterior rim of the ascending rami. On the one hand the H.H. type of the left side elongates the horizontal ramus by its mass production of bone in the angle area and on the other hand the probably existing H.E. influence of the right side pushes towards the left H.H. side.

There is also a difference in length of the ascending rami. The left ascending ramus is 26 mm longer than the right, also being a product of the H.H.

There is a clear reduction in height of the right horizontal ramus in the posterior molar region. The angles measure on the OPT tracing 128° on the elongation side and 104° on the H.H. side. But on the lateral cephalogram, both angles measure differently: 134° on the right side and 115° on the H.H. side. These are the real values. As mentioned earlier, angle values gained by measuring them on the OPT tracing may be wrong but still comparable between the two sides.

The TMJ tomograms (Fig. 74j,k) show a very pronounced enlargement of the left condyle with a slight anterior “beak” and some central areas of sclerosis and translucency. The enlargement also seems to include a bit of the upper part of the condylar neck while the rest of the neck is rather long with a broad base. The glenoid fossa is so steep that it and the condyle do not seem to fit together. The condyle on the right is within the normal range of shape though on the smaller side. The length of the neck is not clearly visible. In the open mouth position both condyles move forward nicely, the enlarged one to almost in front of the articular eminence. These TMJ tomograms do not give enough information. TMJ-ramus tomograms would give much better information.

The p.a. projection of the mandible in the open position was missing.

The lateral cephalogram showed the usual height discrepancy between the two hemimandibles and the front teeth in a slight class III situation.

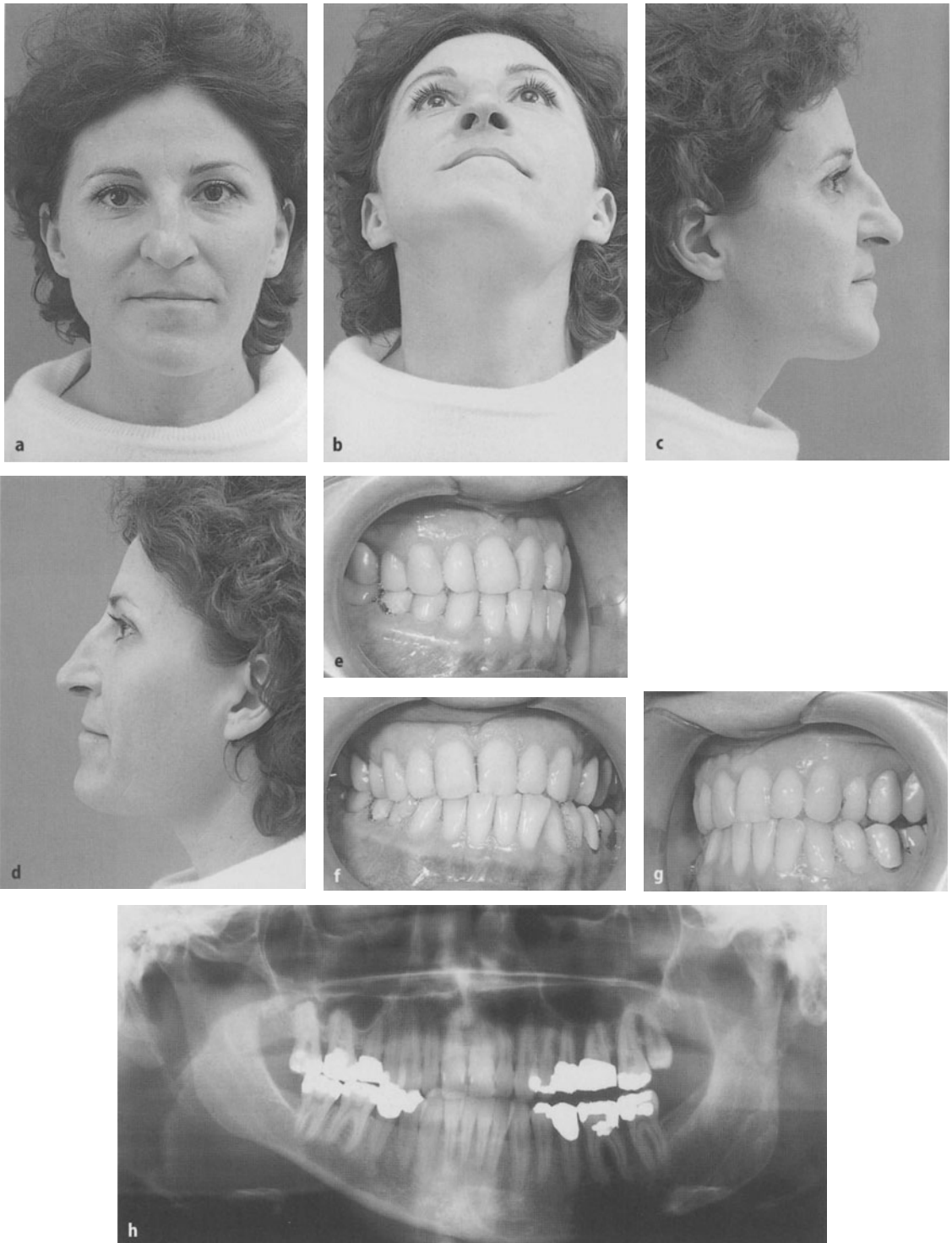


Fig. 74a-h. Unilateral questionable hybrid form and contralateral H.E., slender type. **a-d** In all photographic projections the face shows clear differences between left and right sides. **e-g** The patient's occlusion showing signs of a H.H. on the left side and of a H.E. on the right side. **h** The panoramic view discloses clearly the two different growth anomalies, H.H. on the left, H.E. on the right side

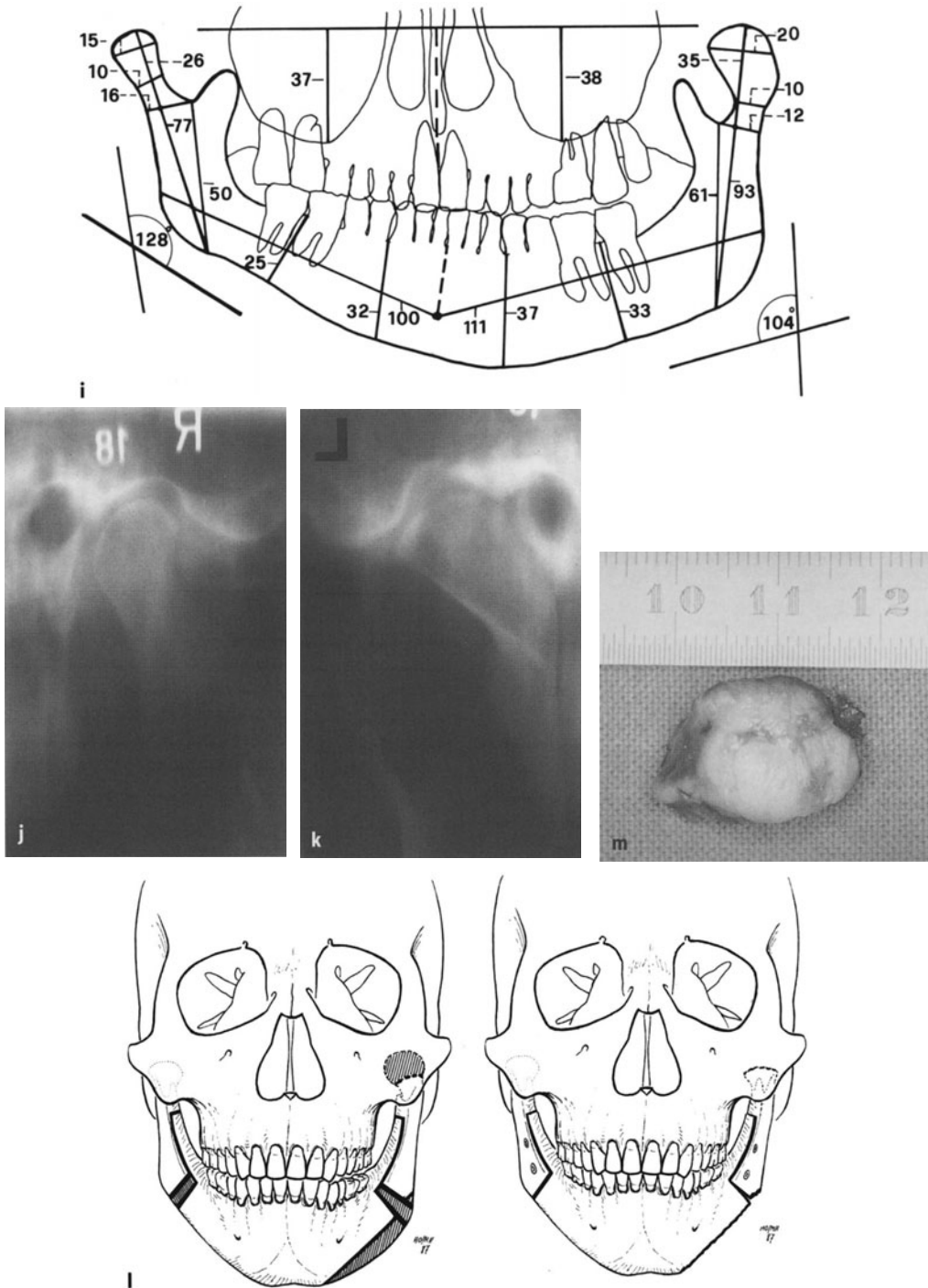


Fig. 74i-m. Unilateral questionable hybrid form and contralateral H.E., slender type. **i** The results of the measurements of the tracing of the panoramic view may give rise to discussion. **j, k** The TMJ tomograms show on one side the typical picture of a H.H. with a small amount of H.E. influence in addition and on the other side a moderate degree of H.E. **l** Diagrammatic illustration of the planned and executed surgical correction. **m** The surface of the resected top of the condyle is clearly nodulated

Provisional Diagnosis. H.H. on the left side and medium degree H.E., slender/non-slender type, on the right side.

Treatment Plan. Intracapsular high condylectomy on the left side, bilateral sagittal splitting of the rami for mandibular repositioning and reduction of the depth of the left body after freeing the mandibular nerve (Fig. 74l).

Actual Treatment. The surgery was carried out as planned, by H. Sailer, then senior coworker at my clinic, using lag screw osteosynthesis. The surface of the resected condyle was found to be nodulous (Fig. 74m). A very good symmetrization of the appearance was achieved.

Histology. The histological appearance disclosed by an older photomicrograph of the resected condyle closely resembled that seen in the specimen from another adult H.H. case (Fig. 32k-m). Splitting of the articular soft tissue and a cleft extending from the articular surface down to the subchondral bone plate were evident. In neighbouring areas the cartilage was in part thin and revealed a deep calcified layer, while in part it was thick and exhibited an irregular osteochondral junction, showing signs of progressive remodelling. Only few marrow spaces were present in the continuous subchondral bone plate.

Diagnosis. Osteoarthrotic condyle lacking any signs of abnormal growth.

Discussion. Although the left side looked, in its clinical and radiological appearance, very much like a typical unilateral H.H., it seemed to me that it also had a small percentage of L-regulator hyperactivity, which would have made it a hybrid form, naturally with the H.H. part more than just predominant. The L-regulator influence had also produced elongation of that side. Two physical signs led me to that diagnosis. One was that the condyle enlargement was not limited to the condyle only but also clearly included the upper section of the neck. The other was that the right H.E. had pushed the chin further over to the H.H. side. As both sides were “fighting” for elongation the resulting occlusion was edge to edge in the front, primarily starting from a skeletal class II situation still noticeable in the panoramic view in the left side molar relationship.

There is another interesting point to be mentioned. The patient had reported that the asymmetry had started at the age of 14 years and finished around the age of 20, but not completely, until she came for surgery at the age of 40. This I doubted, because of the fact that the mandibular canal on the H.H. side was completely down to the lower border of the body. We have learned from other cases that the mandibular canal does run remarkably distant from the lower border when the H.H. develops slowly over 10–20 years. Of course, someone may call this guesswork. I agree, as I would have proof only if a scintigraphic investigation had proven the absolute lack of activity in the enlarged condyle.

This again is a case which teaches that standardized documentation and investigation should be carried out, independent of the growth situation of the individual case.

What are the arguments for the existence of a H.E. on the right side since its horizontal ramus was shorter than that of the other side? The length of the horizontal and ascending rami of the right side were in severe competition with the very strong H.H. of the other side. But there were two points which indicated to me that there was a H.E. on the right side: the reduction in height of the body in the preangle region and the stretched angle. We normally find the reduction in height of the horizontal ramus in the preangle area to be due to a H.H. on the contralateral side and also due to a H.E. on its own side. So that was no proof in this case as to the cause. Another very important sign was the stretching of the angle on the H.E. side. Although on the OPT tracing it measured only 128° only, on the lateral cephalogram it measured 134°, clearly stretched. The angle of the H.H. side measured 104° on the tracing of the panoramic radiograph and on the lateral cephalogram 115°. This means very clearly that looking at the panoramic radiograph and measuring its angles could produce wrong results. As we have standard values for the angles measured on cephalograms, an angle measurement on the OPT tracing is not accurate enough. But 134° measured on the lateral cephalogram permitted me to state that this was a stretched angle indicating a H.E., slender type.

Conclusion. There is always the possibility of encountering an unilateral H.E. and a contralateral H.H., or even a hybrid form. The best therapy in these cases is the early removal of the upper section of the condyle of the site of the abnormal growth. This case also emphasizes the necessity for standardized routine investigations and radiographs.

Hemifacial Hyperplasia

This growth anomaly is always congenital and mostly a part of a generalized hemihyperplasia corporis. The aetiology is not known. If the complete half of the patient's body is involved, then the extremities and corpus, bone as well as soft tissues, are larger and more voluminous than those of the other side. This is, of course, also true for the facial bones, soft tissues and the teeth. The abnormal growth comes to a standstill with the general cessation of growth of the patient.

It is understandable that the patient and his parents seek help before puberty. One can only correct visible

abnormalities, that means reducing the size of the affected mandible, the maxilla, the zygoma, the musculature and also the very voluminous subcutaneous tissues.

As we operate during the growing age, our primary result is always spoiled by the subsequent additional growth, whether it is production of bone mass or soft tissues. The corrective surgery of such a case is not very satisfying in spite of the fact that quite a bit of improvement can be achieved finally, but at the expense of having perhaps to correct the same area twice. The following case does show these difficulties.

Congenital Hemifacial Hyperplasia (Fig. 75)

Sex and Age. Male, 11 years and 9 months.

Aetiology and History. Even in the first weeks of life, a slightly larger right side of the face was noticeable, but it was not so severe that something had to be done urgently. Also, the boy's right thigh was longer than the left and his right foot was larger than the normal left side. His paediatrician diagnosed a "hypertrophy" of the whole right side of the body. When the boy was 4 years old he was referred to an orthopaedic surgeon because his right thigh was 5 cm longer than the left. This surgeon shortened the leg and a plastic surgeon tried to reduce the volume of the right cheek by injecting some sclerosing material, followed by a face-lift type of operation. At the age of 11 years he was referred for orthodontic treatment because of occlusal problems. The orthodontist then sent the boy to me for treatment planning and discussion with the parents of possible corrective surgery to the boy's face. There was nothing relevant in the family history.

Clinical Findings. There is a striking difference in size between the right side of the face and also the right lower extremity compared with the contralateral side. Undoubtedly, the left side is the normal one while the right side is the abnormal half (Fig. 75a). This difference is particularly striking in the patient's face. The right leg also is a few centimetres longer than the left and the right foot and toes are larger than those on the left. The lower leg and the thigh of the right side are much more

voluminous than on the left. The right side of the chest and the right arm, hand and fingers are equal to those of the other side. There is no difference between the right and left sides of the neck.

The whole right side of the face is remarkably more voluminous than the left side (Fig. 75b–d). The increase in volume seems to be both soft tissue as well as bony in origin. The right side of the forehead is higher and more protuberant than the normal left side. The right zygoma with the lateral and infraorbital rim and also the mandible are increased in volume. This is also true for the canine fossa region, clearly pushing the right mandible further down than the left. The chin prominence is divided, with its right side further down and more prominent than the left. The lip commissure is oblique, with the right corner further down than the left and the right eyelid commissure is tilted upwards on its lateral side. The soft tissue is normal on the left side, but very voluminous on the right side of the face. There is less visible vermilion of the right upper lip and more on the right lower lip than on the left side. There is a broad scar of a typical previous face-lift incision on the right side of the face. The right ear is clearly larger than the left, but has exactly the same configuration. Mouth opening is unimpeded but with deviation to the normal left side. Within the mouth, the difference between the left and right sides is also striking (Fig. 75e–h). The right side of the maxilla is more voluminous and it extends higher and

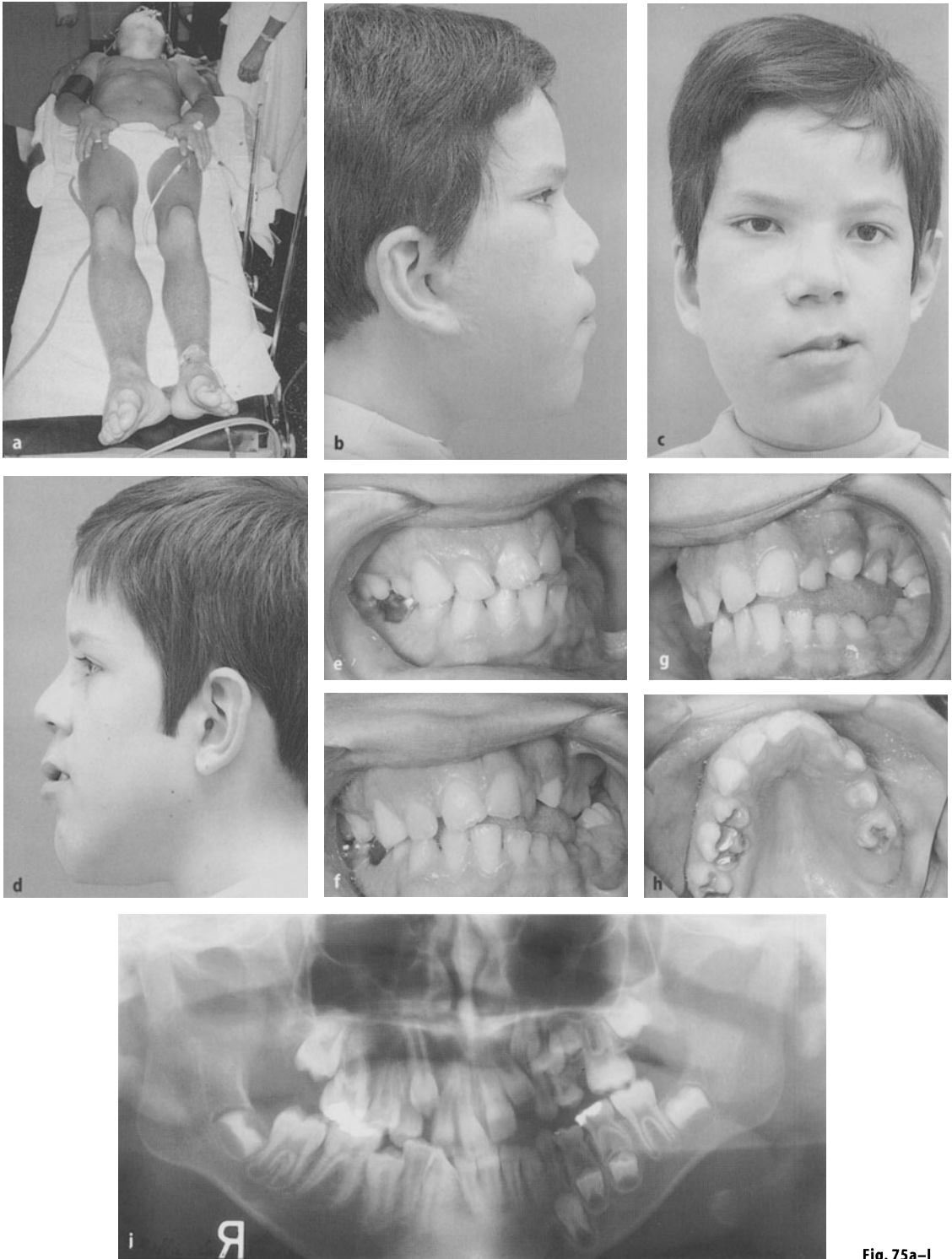


Fig. 75a–i

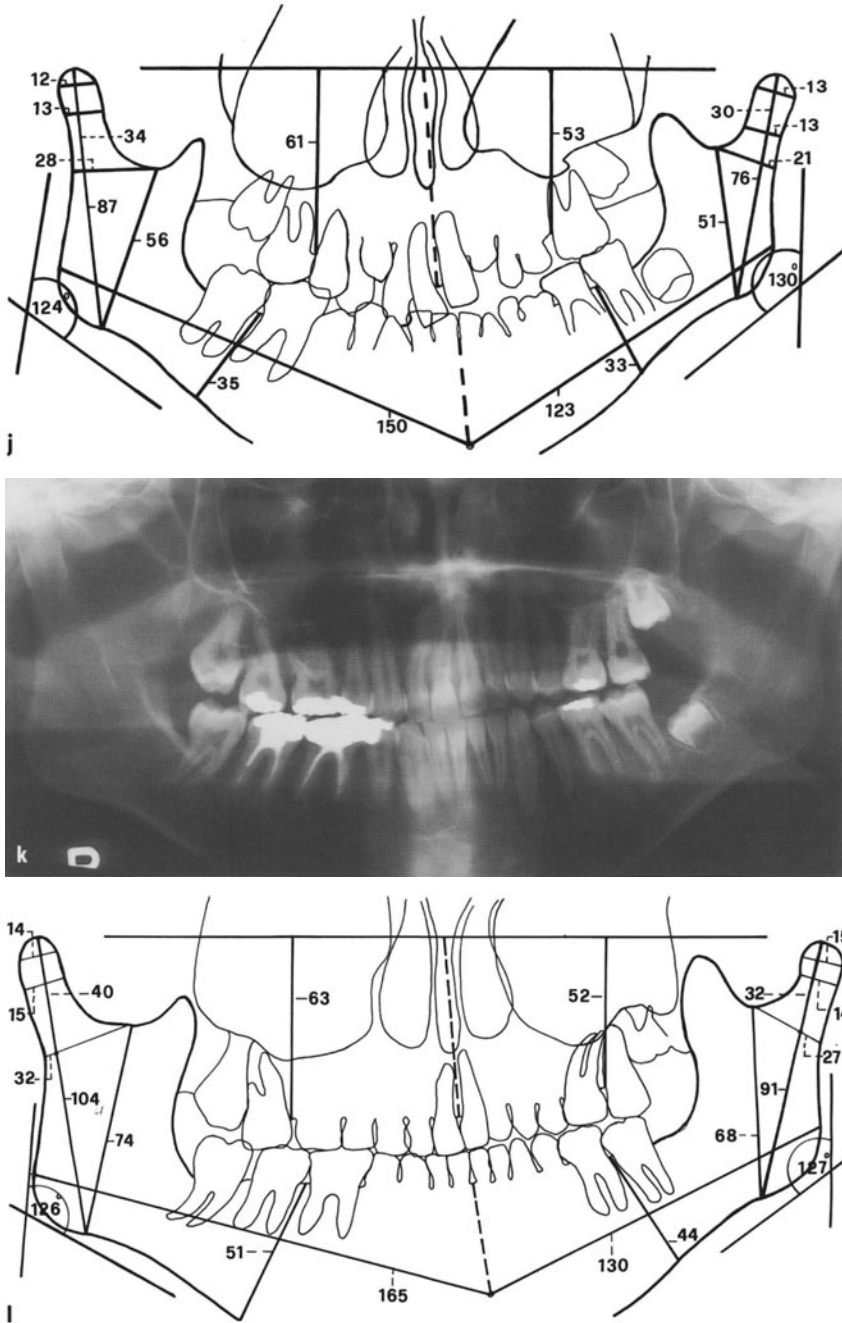


Fig. 75a-l. Congenital hemifacial hyperplasia. **a** Overall view of the patient with his partial hemihyperplasia corporis. **b-d** Facial views of the patient at age 11. **e-h** Occlusion and maxilla mirror view disclose the difference in size between the two sides of the maxilla and of the teeth. **i, j** Panoramic view, taken at 7 years of age and its tracing with the measurements. **k, l** Panoramic view, taken at 14 and 9 months, and its tracing with the measurements

further inferiorly than the left side. Its teeth are fully erupted and larger than the left ones. On the right side one bicuspid is missing while on the left side only the two incisors and the first molar are fully erupted. There is an acceptable occlusal situation on the higher right side in a class III position and an open bite on the left side. The occlusal plane is oblique, descending on the right side parallel to the lip commissure.

Radiographs. *Panoramic view:* At the age of 7 years and 3 months (Fig. 75i) there seems to be no striking difference between left and right sides at first glance. But as soon as details of both sides are compared it became obvious that the right side has larger teeth than the left side, particularly in the lower jaw. The teeth on the left side are not small at all. All the right-sided teeth are almost fully erupted except for the wisdom teeth. The left ones are still deep within the maxilla and the mandibular body with the exception of the first molars. The right side of the maxilla and the right zygomatic arch are further down than on the left, and the right condylar neck is slightly longer and thicker than on the left as well as the whole right ascending ramus. The right coronoid process does not seem to be particularly larger than the left one.

The measurements of the tracing of this panoramic view (Fig. 75j) discloses that all sections of the right mandible are larger than those on the left side and that the difference is quite remarkable. The length of the horizontal rami differs by 27 mm and the ascending rami differ by 11 mm.

At the age of 12 years, the difference in volume of the various parts of the two sides of the face became even more evident. The right wisdom tooth was almost fully erupted.

On the *panoramic view at the age of almost 15 years* (Fig. 75k), the maxilla is now much further down than on the left side, producing a clearly oblique occlusal plane. The degree of difference between left and right sides of the mandible seems to have markedly increased, in the ascending as well as in the horizontal rami.

The measurements of the tracing of that radiograph give clear indications of the differences (Fig. 75l): the difference in vertical height of the ascending rami has not changed much, from 11 to 13 mm. The horizontal rami now differ by 35 mm compared with 27 mm 5 years earlier. Very astonishing is the fact that there is now also a clearly recognizable increase in the difference in the maxillary height. Now it measures 11 mm compared with 8 mm in the previous OPT.

I am aware of the fact that both of these radiographs are very difficult to measure precisely, as the radiographs do not include the full chin area. Regrettably, none of the panoramic radiographs taken later can be used for proper measurement evaluation, as the man-

dible is not sufficiently visible on any of them. Whether this is an effect of bad projection, or technical distortion of the mandible, is very difficult to say. In all the later OPTs the horizontal rami look very high (deep). I can only say they were very high (deep) as I have excised several strips of bone for height reduction and then found it to have increased again.

The condyle and its neck was more enlarged and almost translucent, similar to those found in hybrid form cases. When the patient was 17 years old the maxilla had come further down again on the right side and the mandible had increased in size and volume on the hyperplastic side in all parts. The right condyle seemed to have grown upwards thus elongating the condyle-neck region and with it the ascending ramus.

The panoramic radiograph taken at age 18 years old (Fig. 75m) discloses that 6 months after rotating the lower half of the facial skeleton upwards on the right side, an almost symmetrical situation of the maxilla and the mandible and horizontal occlusal plane has resulted.

The panoramic view taken at age 29 (Fig. 75n) reveals that an enormous amount of bone has developed between the roots of the teeth and the lower border of both horizontal rami, on the right side more than on the left, and also in the whole right ascending ramus.

The TMJ-ramus tomograms (Fig. 75o, p) taken at the age of 17½ years show impressively the enormous height difference between the two sides and a clear but only moderate enlargement of the right condyle-neck section compared with the left.

The p.a. projection of the mandible in the maximum opening position at age 15 (Fig. 75q) demonstrates clearly the difference in vertical height of the mandible and the maxilla on the hyperplastic side. Very typical of a H.H. or a severe hybrid case is the deviation of the mandible to the other side and the lingual tilting of the teeth of that side. Also typical is the separation of the chin into two prominences, the hyperplastic side being greater than the other. But lateral rotation of the contralateral mandible also exists to a minor degree, just enough so that the left mandible looks more voluminous than the hyperplastic side. There is also a clear enlargement of the nasal cavity and the maxillary sinus on the hyperplastic side in this hemifacial hyperplasia case.

The lateral cephalogram taken at the age of 15 years (Fig. 75r) shows a rather similar bilateral height difference between the two halves of the mandible as seen in H.H. cases, however, there is no downward convexity of the lower border on the hyperplastic side.

The maxillary sinus projection in Water's view at the age of 21 years demonstrated clearly the tremendous difference in bony mass in the zygoma region and in the size of the paranasal sinuses.

The full skull p.a. projection disclosed the gross difference in all parts of the right facial skeleton.

Regrettably, *scintigraphy* was not requested.

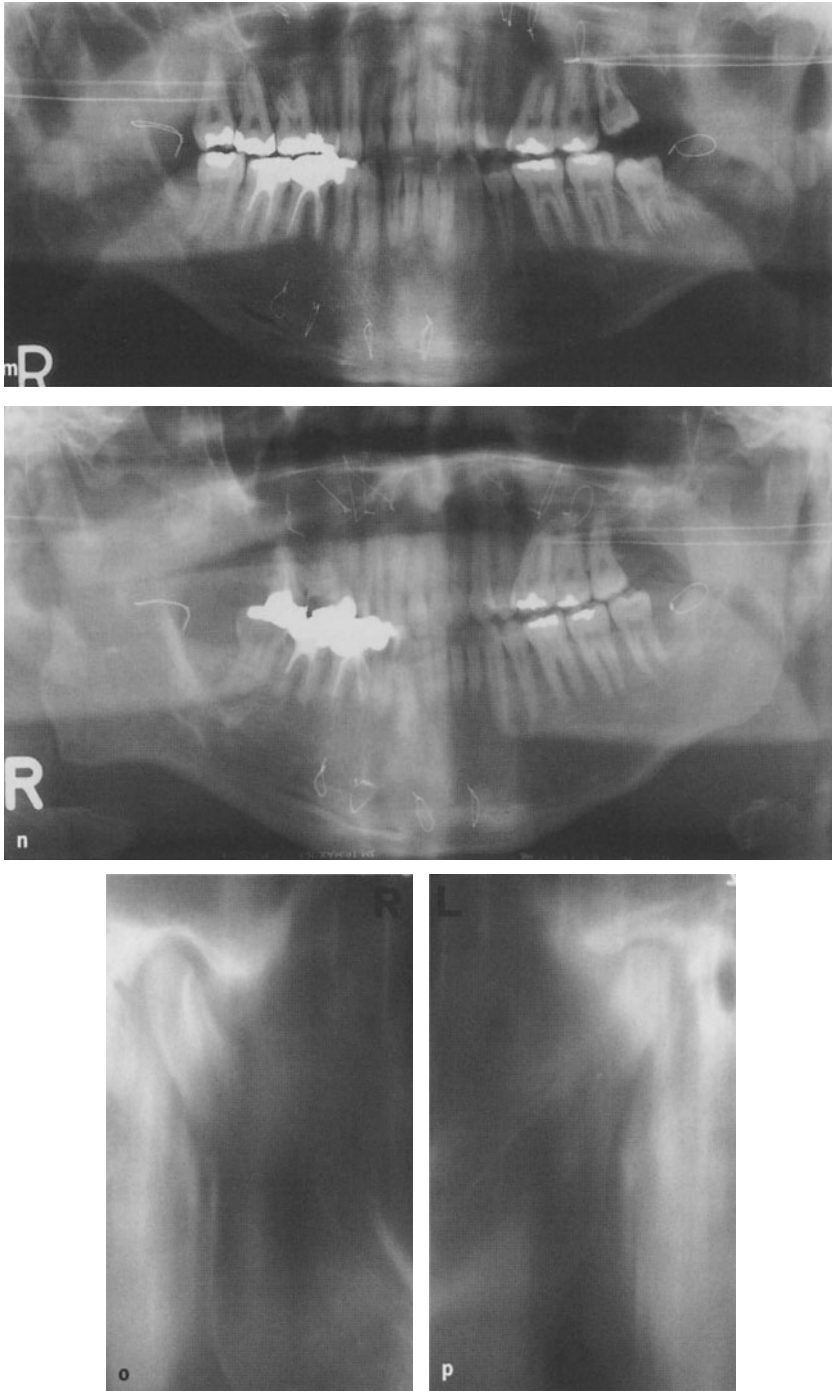


Fig. 75m–p. Congenital hemifacial hyperplasia. **m** Panoramic view, taken at age 18, 6 months after rotation of the lower half of the facial skeleton and reduction of the ascending ramus length and right side body height. A nearly symmetrical maxilla and mandible was achieved. **n** Panoramic view, taken at age 29, discloses that a large amount of bone has developed again in the ascending and horizontal rami of the hyperplasia side as well as in the left horizontal ramus. **o, p** The TMJ ramus tomograms, taken at 17½ years, show the degree of difference in size between the two ascending rami

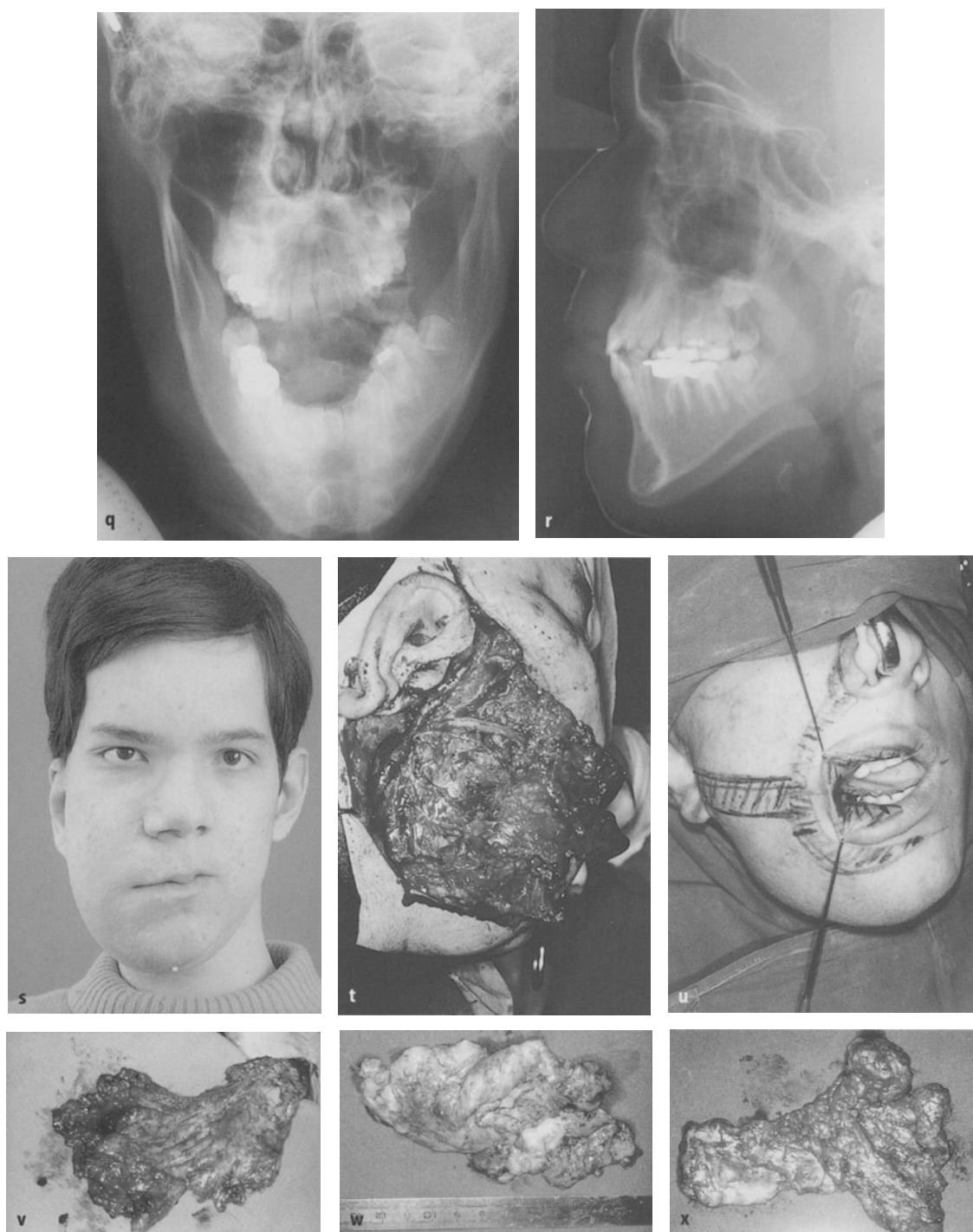


Fig. 75q–x. Congenital hemifacial hyperplasia. **q** The p.a. projection of the open mandible also discloses clearly the difference in size of the right side of the mandible and the maxilla, at age 15. **r** Lateral cephalogram at age 15: clear difference in height of right and left mandibles. The inferior borders show no downward convexity. **s** At age 18 there is still severe facial asymmetry due to a large surplus of soft tissue volume although the skeletal and occlusal situation has become acceptable after rotation of the lower half of the facial skeleton and excision of surplus bone (see Fig. 75m). **t** Subtotal parotidectomy and skin excision for reduction of soft tissue volume. **u** Four months later, surplus skin in the cheek and around the mouth was excised together with fibrotic tissue. **v–x** Within the following 1 1/2 years, on three occasions regrown subcutaneous tissues were excised

Provisional Diagnosis. Hemihyperplasia of the face in a case of incomplete hemihyperplasia corporis.

Treatment Plan. Combined orthodontic treatment and surgical correction of the bony and soft tissue surplus of the affected half of the face has to be performed in accordance with the development of the anomaly.

Actual Treatment. The surgery became the most important part in the actual treatment. The orthodontic treatment was, for some years, done in close cooperation with the necessary surgical interventions. After some time the patient refused further orthodontics. At the age of 15 years and 10 months the surplus of the lower border and chin on the hyperplastic side was reduced. The right lower vermilion was partially excised. When the patient was 17 years and 7 months, the lower half of the facial skeleton was rotated to the right and upwards, using the author's facial rotation procedure. By this procedure the whole right side was reduced in height. A bilaterally equal height was achieved together with a horizontal occlusal plane. The soft tissue volume was now the greatest problem (Fig. 75s). Half a year later, the soft tissue volume of the cheek had to be reduced. A conser-

vative parotidectomy and a facelift were performed (Fig. 75t), and 4 months later it was found necessary to excise all the cheek fat and fibrous tissue in addition. Moreover, quite a large strip of skin, in the vertical dimension as well as around the mouth, had to be removed (Fig. 75u) in order to obtain more tension and flatness in the cheek area. The patient was now 19 years old. Half a year later, the volume of the upper and lower lips was reduced again. It seemed as if the soft tissue volume would continue to increase the more it was excised. When the patient was nearly 20 years old, the right upper and lower lips had to be thinned again and the right lip corner was suspended by the insertion of a fascia lata strip. At the age of 21 $\frac{1}{2}$ the maxilla had to be repositioned for a second time to the left side and upwards. It had grown downward and to the right. Once more the fatty tissues of the right side of the face had to be thinned. This was the third time this had had to be done (Fig. 75v-x).

After the last repositioning of the maxilla at age 21 $\frac{1}{2}$ there was not much further production of soft tissues. The skeleton had not yet stabilized, although its additional growth was not that severe. It seemed to slow down and stopped around the age of 23 years. The bone

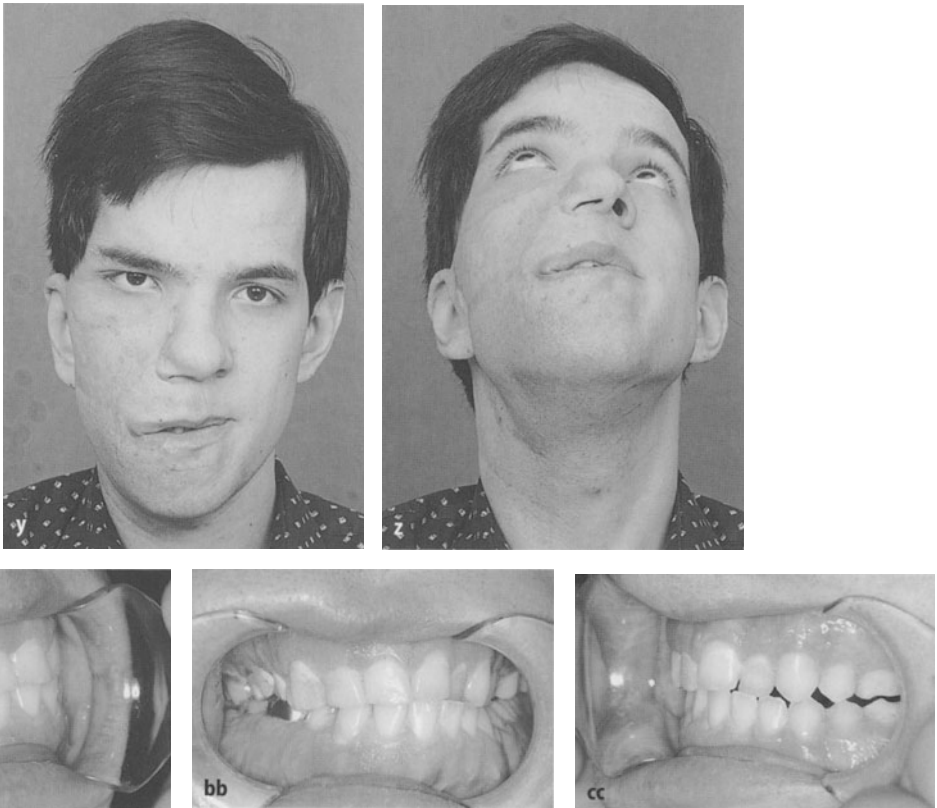


Fig. 75y-cc. Congenital hemifacial hyperplasia. **y, z** Patient's appearance at age 23. **aa-cc** Patient's occlusion at the same age

production had involved the right maxilla and the ascending as well as the horizontal rami, producing again some obliquity of the occlusal plane. There was a large increase in height of the right side, but also in the height of the left horizontal ramus (see Fig. 75m). The result was somehow acceptable in the patient's appearance (Fig. 75y, z) as well as in the occlusion (Fig. 75aa–cc).

Now that all tendency to abnormal tissue production, bone as well as soft tissues, had ceased, it would now be possible to improve the facial and occlusal situation even more and probably for good.

Discussion. Hemifacial hyperplasia as part of a hemihyperplasia of the whole body reveals, of course, not only different clinical and radiological findings in the facial skeleton but it also includes hyperplasia of all the soft tissues, the sinuses, the teeth, and even the gingivae. And what makes it even worse is the fact that its growth capacity behaves very unfavourably, differently in both the skeleton as well as the soft tissues. If one looks at the mandibular size and shape, only a certain similarity to a hybrid form of H.H. and H.E. could perhaps be recognized as when both condylar growth regulators participate almost equally. But still, the severe rotation and rapid downward growth are missing and also the very important downward convexity of the lower border of the mandible. While we believe we know the growth steering mechanism of H.H. and H.E. and its hybrid form anomalies, there is nothing similar that we could identify as being responsible for the continuing increase in bone and soft tissue mass of a true hemifacial hyperplasia case.

Very regrettably, in those days I did not think of a high condylectomy. From two points of view it would have been very interesting, the behaviour of further growth of that side of the mandible and the histology. However, I am of the opinion that the bone production

mechanism in such a case differs entirely from the condylar hyperactivity type. It must be so, otherwise the maxilla and the zygoma would not have been influenced.

In this case, the growth activity seemed to have come to a standstill at the age of 23 or so. Still, the soft tissue problem was much worse than that of the facial bones. There seemed to be production of subcutaneous fibrous tissue on and on, so that even the soft tissue volume loss due to a parotidectomy was soon replaced by new fibrous soft tissue. The whole anomaly is very interesting from the pathophysiology point of view.

I would like to add some remarks regarding the terminology. The same condition is sometimes called hemifacial hypertrophy in a case of hemihypertrophia corporis or it is called hemifacial hyperplasia. Hypertrophy means volume enlargement through enlargement of the individual cells of the organ. Hyperplasia means enlargement due to an increase in cell production which leads to the enlargement. This case was surely a hyperplasia because whatever I resected, bone or soft tissues, grew again in even greater mass by production of the tissues. This cannot happen due to the increase in the size of the individual cells but only through cell multiplication.

Conclusion. True hemifacial hyperplasia which is a part of a hemihyperplasia of the whole body is a unilateral growth anomaly by itself, not comparable with any other mandibular growth asymmetry. It is not only pathophysiologically more difficult to understand than those due to condylar growth misregulation, but also therapeutically much more difficult and less satisfying to deal with. Based on the experience with this case, it might be best to postpone the surgical correction, at least the correction of the soft tissues, until the surplus growth has completely ceased.

Mandibular Growth Anomalies in Acromegaly

When lecturing on the subject of mandibular growth anomalies I have often expressed the opinion that growth hormone overproduction in an adenoma of the pituitary gland will stimulate the condylar growth factors M or L, or both. So, in acromegaly, bilateral mandibular growth anomalies equal to bilateral symmetrical H.E., H.H. or hybrid forms will be produced. This was, in my opinion, the reason why in acromegaly only the mandible can increase in length and mass producing a rather typical antemandibulism type while no other bones of the human skeleton will become longer as they do not contain anything similar to our assumed condylar growth factors.

As we did not know enough about the reaction of the various bones to the overproduction of human growth hormone, I asked two students to find the relevant information on the available number of patients with acromegaly. They found 31 patients with acromegaly, proven by overproduction of human growth hormone, in their investigation. A. Künzler (1988) had the task of obtaining as much information about the influence of acromegaly on the viscerocranium. B. Degiacomi's (1991) duty was to find out what kind of changes in the neurocranium accompany acromegaly. Both doctorate theses are very interesting.

For the subject of mandibular growth anomalies, the findings of the influence of the growth hormone on the viscerocranium is of practical interest only, particularly its influence on the mandible. Künzler compared the results with those found in a group of 25 control subjects. There were only 6 patients out of 31 proven acromegaly cases who developed an antemandibulism and in all 6 cases this was symmetrical. They had come to our clinic for corrective surgery. He concludes that "There are distinct alterations in the mandible, in the whole length of the ascending ramus as well as in the horizontal ramus, in particular in the chin prominence". He expresses the opinion that both types of condylar hyperactivity (H.E. and H.H.) are also found in acromegaly. He also states that "aside from these form anomalies there are also pathological mandibular configurations which cannot be attributed to these two growth anomalies".

When I tried to evaluate these cases of acromegaly-induced mandibular growth anomalies I found that radiologically they may look like a bilateral symmetrical H.E. or H.H. form but they lack the corresponding condylar forms.

The fact that only about 20% of acromegaly cases produce real antemandibulism and that these are of a H.E. or a H.H. type but without the corresponding condylar forms, leads me to the conclusion that in acromegaly, the growth stimulation is not the same as in the pure condylar hyperactivity cases. Three cases will demonstrate this.

If this whole philosophy of the existence of condylar growth regulators is of any value in the understanding of various typical mandibular growth anomaly types, or even accepted as a fact, then we should not be surprised to find one or other of these typical anomalies also developing in a case of true acromegaly. This has been reported by H. Müller in 1979, in a case where the patient developed acromegaly and simultaneous mandibular asymmetry at the age of 23. According to his illustrations there was no doubt about the acromegaly. But I cannot see from the radiograph whether it was a H.E., H.H. or a hybrid type of mandibular asymmetry. From the patient's face I would diagnose a hybrid type of condylar hyperactivity as being the cause, as there is clear deviation of the mandible to the other side and clear increase in facial vertical height and flatness on the affected side.

H. Müller discussed the possibility of the hormonal influence for the simultaneous development of the two growth anomalies, acromegaly and the unilateral condylar hyperactivity, then still called "condylar hyperplasia". He states that "an aetiological relationship between the hormonal dysfunction and condylar hyperplasia is not proven". According to my experience and knowledge, the two different mandibular growth anomalies cannot have the same hormonal cause, for three reasons. In the great number of acromegaly cases this anomalous combination should occur more often. H. Müller's case is the only one so far reported. Secondly, those mandibular anomalies that I have seen as a result of acromegaly do not fit into the anomaly types found in condylar hyperactivity. Thirdly, condylar growth regulator hyperactivity can start at any time from 5 years of age up to 50 years or probably even more. I would therefore classify this case as a result of a very rare occurrence of having these two different growth anomalies in one mandible. Even in the 66 cases of M. Rushton (1951), he does not mention a case of simultaneous appearance of "condylar hyperplasia" and acromegaly.

Acromegaly of Bilateral Pseudo-H.E., Slender Type (Fig. 76)

Sex and Age. Male, 56 years of age.

Aetiology and History. Some years previously the patient had realized that his facial appearance had changed. He did not remember how long back. When he went to see his general medical practitioner he was referred to an endocrinologist who diagnosed a surplus of growth hormone production. Therefore, he was referred to a neurosurgeon, who operated on his pituitary gland tumour. As he had a full upper denture and the remaining lower teeth were placed in front of it, his dentist had informed him that without a set back of the mandible there was not much chance of a better-functioning upper denture. He now sought more detailed information about what ought to be done to his jaws.

Clinical Findings. The patient is of rather large build. His head is broad and rather round (Fig. 76a,b). His lower lip protrudes, but not his chin. The soft tissues are very thick. It is impossible to gain an idea of the shape of the angle areas. Mouth opening is normal.

The lower front teeth protrude with lots of space between them. There is no occlusion with the full upper denture (Fig. 76c–e). The tongue is very large. The maxilla is edentulous with a badly-fitting full denture.

Radiographs. The panoramic view (Fig. 76f) reveals a mandible of unusual shape. Both sides are absolutely straight, from the condyles to the chin. On the left side there is no angle at all and on the right side just a very rudimentary one. Both hemimandibles are rather slim, with most of the cheek teeth missing. The condyle looks just like a cherry sitting on top of a thin straight stem.

The TMJ tomograms were regrettably missing.

The lateral cephalogram (Fig. 76g) shows the very classical facial skeletal changes typical of acromegaly including a prominent forehead due to extensive pneumatization of the frontal sinuses. The mastoids are overpneumatized and the sella turcica is very large. The ethmoid cells also look expanded. The base of the max-

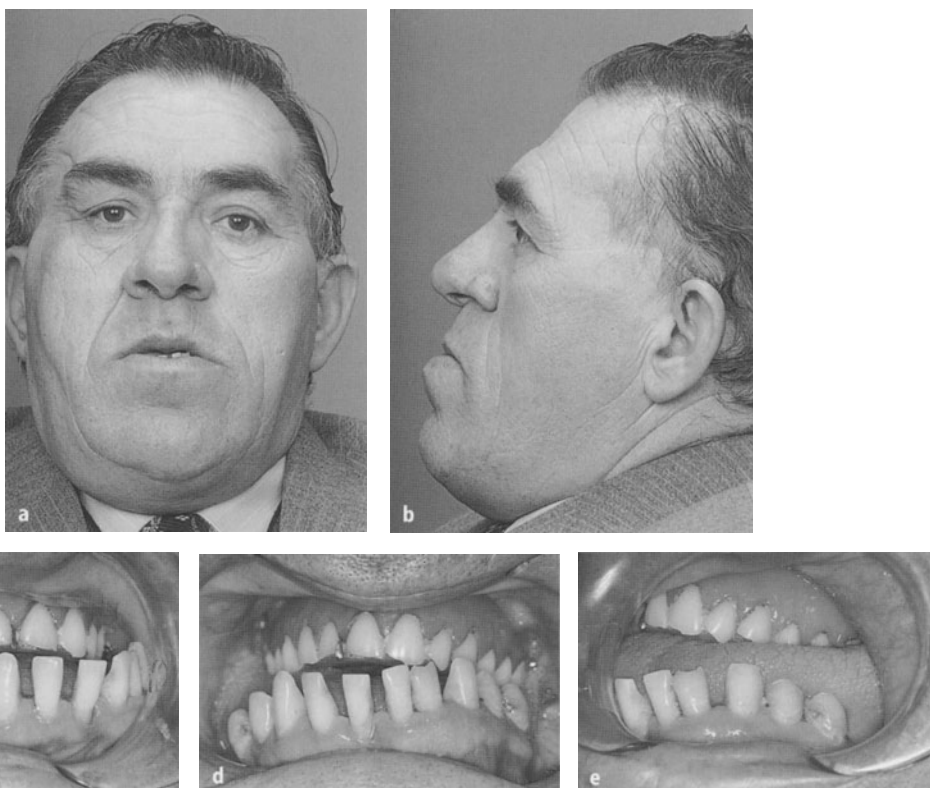


Fig. 76a–e. Acromegaly of bilateral pseudo-H.E., slender type. **a, b** The patient's appearance does not permit any conclusion regarding the shape of its skeletal background. **c–e** The full upper denture has no occlusion with the remaining mandibular teeth. The latter are protruded with large interdental spaces due to a macroglossia

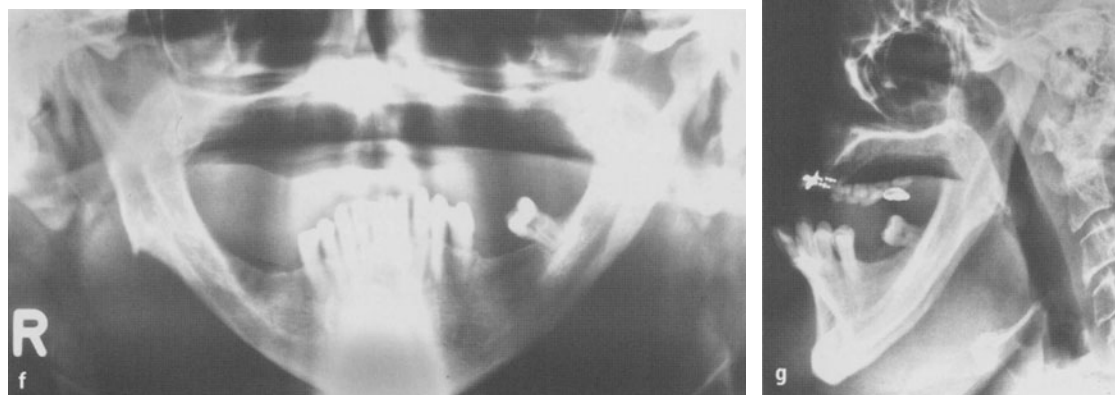


Fig. 76f, g. Acromegaly with bilateral pseudo-H.E., slender type. **f, g** The panoramic view as well as the lateral cephalogram disclose a slim and straight mandible with thin necks and rounded condyles

illa is on the short side and was too far back (retromaxillism). The mandible was straight like a stick, from the condyles to the chin. The chin itself does not protrude very much, but the remaining front teeth are tilted forwards.

Provisional Diagnosis. Acromegaly with an elongated mandible of the bilateral H.E., slender type, and retro-maxillism.

Treatment Plan. In order to create a normal intermaxillary relationship and a favourable situation for a subsequent denture, the following surgery has to be carried out: advancement of the maxilla by a Le Fort I-type osteotomy and a set back of the mandible by bilateral sagittal splitting of the rami with the lateral osteotomy extending to the angle, in order to rotate the body into a horizontal position. In addition, the protruded anterior alveolus with its teeth should be brought into an upright position. In order to prevent relapse of the repositioned alveolar segment, the size of the tongue must be reduced.

Actual Treatment. The patient refused any surgery of the type suggested.

Discussion and Conclusion. In this case of acromegaly, the mandible had a very similar shape to that observed in cases of severe bilateral H.E., slender type. In the latter case one would not expect such very round medium-sized condyles but rather condyles of the same width as their necks. It is interesting to note that the mandible

does not seem to have gained much in length, with this change mainly through the increase in the angulation. The enlarged tongue had gained sufficient space by pushing the lower teeth forward. Because of this it would be essential to reduce the size of the tongue before the protruded lower alveolus was repositioned, in addition to the set back and upward rotation of the mandible. I have learned this through experience gained in some cases which led me to the following conclusion: *“The active cause of a facial skeletal anomaly is stronger than screws and plates. Plates and screws cannot prevent a relapse if the cause of the anomaly is still active”* (Principle No. 31).

This case gives reason enough to especially mention that the external appearance of an antemandibulism case does not always permit conclusions regarding its skeletal background type. The patient had a very round face and a rather small negative overjet and yet both hemimandibles were straight like sticks. When we look at paintings from the various ancient members of the famous Habsburg family we generally see two different facial types of their antemandibulism. In one type they have a very slim long face with a pointed chin. In these individuals we would expect a similar type of mandible as in this case. But there is also the other type of the Habsburg jaw with a round face. But since we do not have any radiographs of these famous antemandibulism cases we cannot know the shape of the skeletal structure of their faces. This case proves, the appearance does not permit one to make a correct diagnosis of the shape of the mandible without radiographs.

Acromegaly of Bilateral H.E., Non-Slender Type (Fig. 77)

Sex and Age. Male, 50 years of age.

Aetiology and History. When the patient was 44 years old he was referred to the Department of Endocrinology in a very large governmental hospital because of his acromegaloid appearance. No such abnormality in growth hormone values was found. Because of this result, no therapy seemed indicated. He himself had never realized that his head, hands and feet had become larger. His dentist had now sent him for improvement of his intermaxillary relationship for prosthetic reasons. We sent him to the Department of Endocrinology at the University Hospital of Zurich. Again the growth hormone values were found to be normal at this very competent second institution. Thereafter, a mandibular push back operation was performed using the bilateral sagittal splitting procedure on the rami. A good intermaxillary relationship was achieved and 4 months later the patient was provided with prostheses. Two years later, at a routine follow up, the mandible seemed to have moved forward by 1–2 mm and 4 years after his mandibular set back operation, his tongue was found to be larger and his lower teeth protruded with larger interdental spaces. His fingers seemed thicker and his mandible had come forward by 3–4 mm. He was again sent for endocrinological screening. Once more the result was negative as far as a growth hormone abnormality was concerned and 4 years later, i.e. 8 years after the surgery on his protruding mandible, the same endocrinology department now found proof of an active acromegaly, but not to such a degree that neurosurgery was indicated. The patient received medication and his intermaxillary relationship was improved by advancement of the edentulous maxilla.

Clinical Findings. The patient presented all the classic facial signs of acromegaly (Figs. 77a,b). His middle third seemed very flat while the very protrusive mandible seemed broad with rather stretched angles. The chin was rounded and the mouth movement normal. The tooth situation was very poor. The first bicuspid, canines and anterior teeth were the only teeth in the mandible. The cheek teeth were missing and also a few of those in the maxilla. There was no occlusion whatsoever and a negative overjet of 14 mm.

Radiographs. The panoramic view (Fig. 77c) showed an almost symmetrical, strong mandible with condyles and their necks of rather large size and shape and there were also rather unusually long ascending rami with stretched angles, more so on the left than the right. The mandibular body on both sides seemed very long and high. The midline of the lower front teeth and the sym-

physis was deviated to the left side by about 4 mm. The mandibular canals ran at a normal distance from the lower border on both sides. The right body was remarkably higher than the left which was also higher than normal, considering the fact that it was edentulous. The trabecular structure of the mandibular bone was rather coarse.

Measuring the tracing of this radiograph was only partially possible as parts of the two condyles were not on the radiograph. The length of the trunks differed by 7 mm, in favour of the right and the horizontal rami differed by 3 mm in favour of the left side. The most striking difference was found in the height of the horizontal rami, the right being 10 mm higher (deeper) in the molar region than the left. Also, the angles differed quite a bit, the left, at 142° being 10° larger than the right. There was nothing to signify any of the condylar hyperactivity abnormalities.

The TMJ tomograms (Fig. 77d,e) show rather large condyles with a partially flattened surface. The articular eminences are flattened and sclerosed. The right condylar neck seems a little elongated but of normal thickness while the left seems thicker and its condyle a little larger than normal.

The p.a. projection with the mandible open showed nothing remarkable other than a very large mandible which deviated a little to the left.

The lateral cephalogram (Fig. 77f) shows the typical acromegalic facial skeleton with severe retromaxillism. The mandible is large and had almost no angles. Also, the supraorbital rims protrude and the nose is very large with a drooping tip. The sella turcica is not clearly defined.

Provisional Diagnosis. Acromegaly with bilateral H.E., non-slender type, deformities with some additional asymmetries of the various parts of the mandible, and retromaxillism.

Treatment Plan. Anterior repositioning of the maxilla and retropositioning of the mandible utilizing a Le Fort I-type osteotomy and bilateral sagittal splitting of the rami.

Actual Treatment. The surgery was carried out by a trainee according to the treatment plan.

Discussion and Conclusion. Within the scope of this book, it is the abnormal growth of the mandible which is the main interest. Naturally, it would also be very interesting to know how, in acromegaly, the various

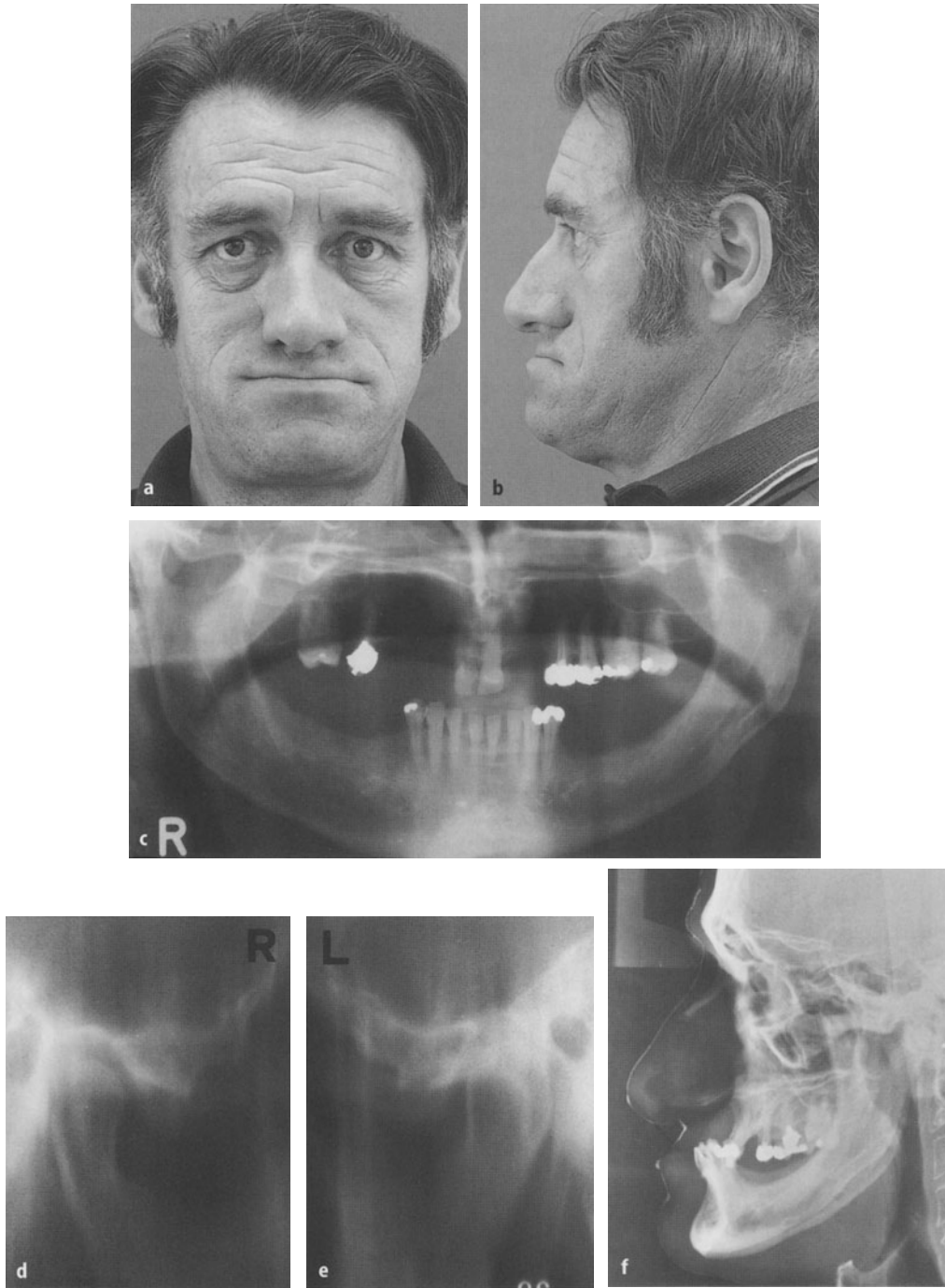


Fig. 77a–f. Acromegaly with bilateral H.E., non-slender type. **a, b** The patient's appearance is very typical of acromegaly. **c** Panoramic view: very large and strong mandible with stretched angles. **d, e** The TMJ tomograms disclose rather large and differently shaped condyles, on the left with a short thick neck and on the right side with an elongated neck. **f** The lateral cephalogram reveals a facial skeleton typical of acromegaly with a large and strong mandible with stretched angles and severe retromaxillism

skeletal parts increase in volume. But that has not yet been determined. However, regarding the mandible, I tend to be of the opinion that the hGH (human growth hormone) output might reactivate one, the other, or both condylar growth regulators thus causing them to produce bilaterally either a H.H. type, a H.E. type or a hybrid form anomaly of the mandible. In this case it is the shape of a mandible which is very much akin to a bilateral H.E., non-slender type, in a primarily rather strong mandible. Although both hemimandibles and both condyles are not absolutely symmetrical in shape, we still have to accept that, in this case of acromegaly,

the mandible has grown to a shape similar to a bilateral H.E., non-slender type. I do not have any other case with the same anomaly of non-acromegalic origin. I would expect absolute symmetry because the anomaly is centrally steered, except if there was already a pre-existing asymmetrical bilateral H.E. before the acromegaly developed. So, what do we really know about these things?

This case also tells us that the clinical signs must always be congruent with the laboratory values. Often enough, it is not the clinical diagnosis which is wrong, but the laboratory findings which were just not sensitive enough to clarify it.

Acromegaly with Bilateral Hybrid Type Condyles, but a H.H. Type Mandible (Fig. 78)

Sex and Age. Male, 38 years of age.

Aetiology and History. The patient had noticed the first signs of acromegaly 5 years previously. His pituitary gland adenoma had been removed 1 year ago. He now sought information regarding possible correction of the facial skeletal abnormalities caused by his acromegaly. He claimed that before the acromegaly developed he had had a normal overbite, with no negative overjet.

Clinical Findings. The patient was of average build. He seemed to have a strong general skeleton with broad shoulders. He had a round face (Fig. 78a,b) with a number of clear signs of acromegaly including a strong and protruding mandible, a large nose, enlarged supraorbital rims and zygomatic bones, thick skin and fingers, etc. His broad chin prominence was deviated slightly to the left. The strong mandible had rounded angles and straight lower borders. The mouth movements were normal.

Almost all teeth were present (Fig. 78c). On closure he had a negative overjet of a few millimetres. The lower teeth midline was shifted to the left by the width of about half a lower incisor. His tongue was rather large.

Radiographs. The panoramic view (Fig. 78d) discloses an extremely strong mandible with both sides almost equal. Both condyles seem to be of the same p.a. diameter as the well elongated necks which are a little on the thick side. On both sides, the ascending rami including the condyles seem clearly longer than normal. They make a strong but not abnormally curved angle with the very high mandibular bodies. The incisor midline is shifted to the left by the width of half a lower incisor. The mandibular canals run rather high on both sides, and on both sides the body has a strikingly strong trabeculation. This is not the case in the ramus and condylar regions.

The evaluation of the tracing of the panoramic radiograph (Fig. 78e) proves that both hemimandibles are al-

most equal. The horizontal ramus on the right side measures 3 mm more in length than does the left. All other measurable distances are almost equal. The right angle is 8° larger than the left, the latter, at 114° is smaller than the average.

The TMJ tomograms (Fig. 78f,g) confirm the existence of rather strong and very long condylar necks and, to some degree, enlarged condyles of abnormal shape. They are not really distinguishable from the neck, as if the enlargement of the condyles has extended into their necks. The right condyle has a small anterior "beak" and the left has on its top a slightly sclerosed, protruding irregularity.

The p.a. projection of the mandible in the open position (Fig. 78h) also demonstrates the very strong mandible with long ascending rami and large condyles. The maxilla too seems rather high in the vertical dimension. Both ascending rami and condyles have a strikingly coarse bone trabeculation.

The sinus projection in Water's view showed very large frontal sinuses and also rather strong zygomatic bones. The maxilla again looked very high.

The lateral cephalogram (Fig. 78i) confirms the typical acromegalic skeletal picture with a rather short and retrodisplaced maxillary base.

Provisional Diagnosis. Macromandibulism and microretromaxillism in a case of acromegaly. The shape of the mandible was almost a bilateral H.H. type but without the necessary inferior convexity of its lower borders. The condylar necks were of the H.E. type with a condyle neck appearance and shape as in mild hybrid form cases.

Treatment Plan. Advancement of the maxilla by 10 mm into a normal overjet position and reduction of the supraorbital rims and nose.

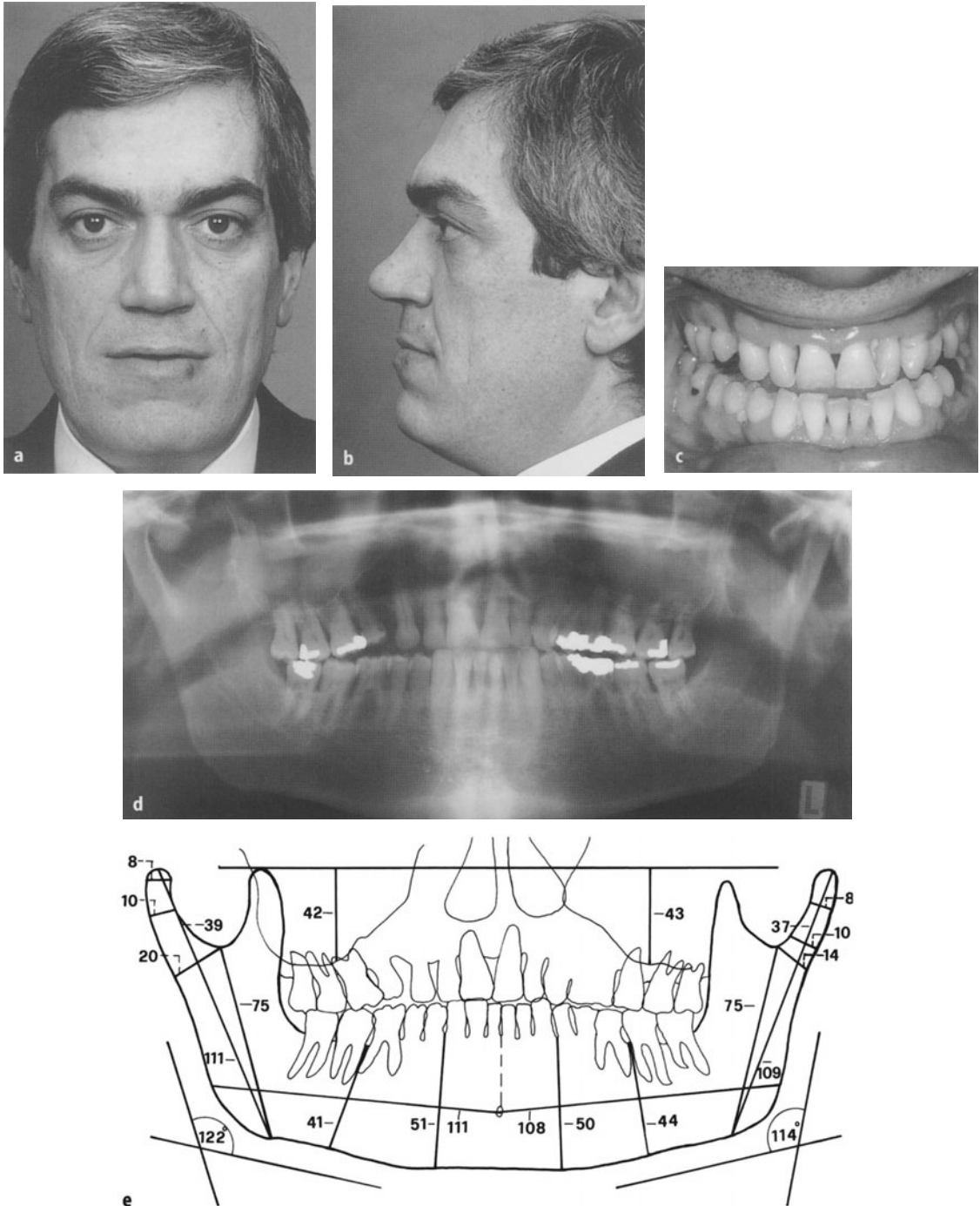


Fig. 78a–e. Acromegaly with hybrid type condyles, but a H.H.-type mandible. **a, b** Typical acromegalic appearance in front and side views with a strong, rounded mandibular shape, no chin deviation. **c** In occlusion there is a negative overjet with the lower midline a little over to the left. **d** The panoramic view discloses a very strong mandible equal on both sides, normal curvature of the angles, but long and rather thick condylar necks with no clear condyles, very similar to the articular process of the H.E., slender type anomaly. **e** The measurements of the tracing disclose that both hemimandibles are almost identical

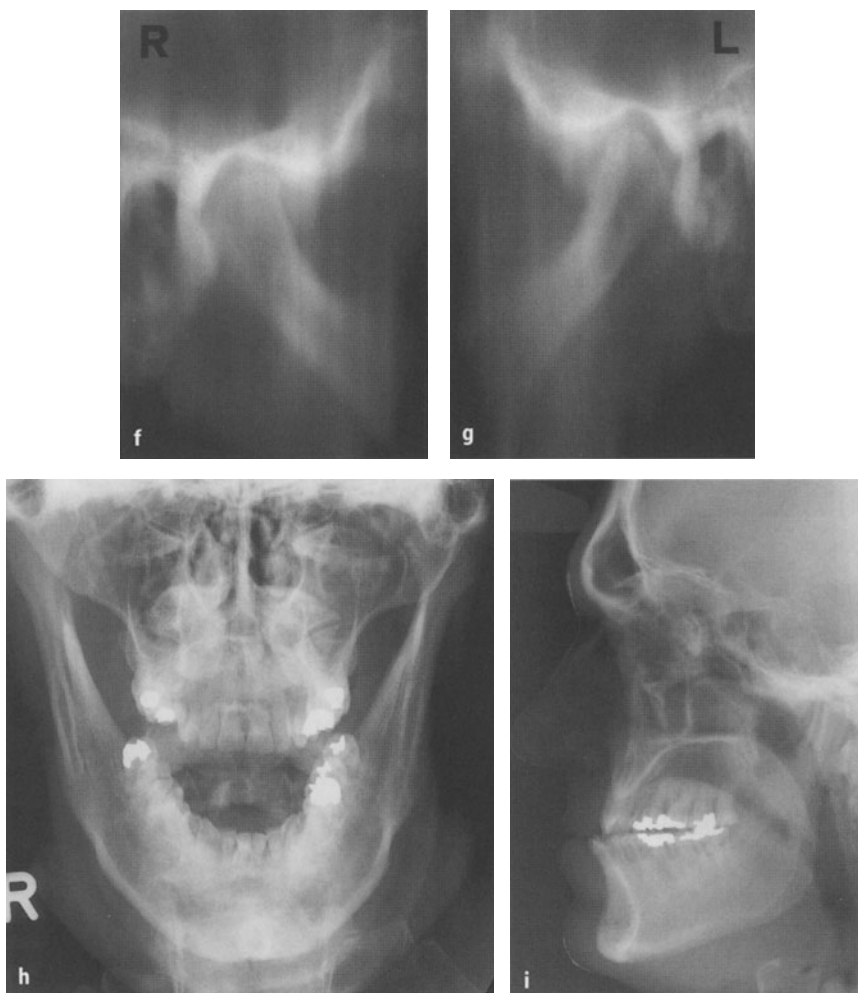


Fig. 78f–i. Acromegaly with hybrid type condyles, but a H.H.-type mandible. **f, g** The TMJ tomograms disclose a neck-condyle situation similar to hybrid forms. **h** The mandible p.a. projection in the open mouth position reveals a strong mandible with long ascending rami and large condyles. **i** The lateral cephalogram shows a facial skeleton which is very typical of acromegaly with a strong mandible without any inferior convexity of its lower rim and, in addition, a medium degree micro- and retromaxillism

Actual Treatment. The patient did not present himself for surgery.

Discussion. The patient primarily probably had a straight retroface profile which is typical of Greek people like himself. Therefore, it seems logical not to reposition the mandible posteriorly, where it had come from and also in order to prevent the necessity of reducing the large tongue, but to advance the maxilla. So the correction of the occlusal problem would have been very easy.

In this acromegaly case, the mandible seemed extremely strong and with an unusual body height and length of the condylar necks. The very much elongated

and thick condylar necks did not match with the almost rectangular strong mandible. These necks along with their irregularly formed, rather slightly enlarged condyles, would normally match those found in a hybrid form with the H.E. very predominant. But then the angles of the mandible should be more stretched. So the whole mandible seems to consist of parts which do not fit together according to our knowledge and understanding of mandibular growth and its misregulation.

Conclusion. In acromegaly, the abnormal growth of the mandible does not follow the pattern of H.E. or H.H. or its hybrid forms.

Histology of Condyles in Mandibular Growth Anomalies

18.1

General Considerations

A survey of the presented cases discloses the marked variability of most types of mandibular growth anomalies, not only with respect to clinical appearance, but also regarding age at onset, duration and age at surgical intervention. Thus, in cases of condylar hyperactivity, i.e., H.H., H.E. and hybrid forms, growth can apparently turn abnormal during the regular growth period, continue beyond the age of regular growth cessation or resume activity during adulthood. Depending on the interval between the start of the disorder and condylectomy, some of the resected condyles may have been growing actively, while in others growth may have ceased. The questions we hoped would be answered by the histological examination were therefore:

1. Was condylar growth still going on at the time of surgery?
2. Did it proceed at a normal rate?
3. Was the mechanism of growth normal?
4. Is there any relationship between the structure of the condyle and the clinical picture of the growth disorder?

As the normal microscopic appearance of mandibular condyles alters during the growth period, after growth has terminated, and finally also during adulthood (H. Luder 1996), the interpretation of histological findings in specimens affected by growth disorders has to primarily rely on age-matched controls. This should allow answers as to whether growth was still active or had ceased. Also, conclusions should be possible regarding the mechanism and timing of growth. However, as will be outlined below, estimates regarding the rate of growth are more problematic. Finally, attempts at relating histological characteristics of condyles to particular types of growth anomalies are impeded by the great variability in clinical appearance and, hence, the limited number of uniform cases.

18.2

Normal Condyles in Different Age Groups

Growth Stage. Throughout the period of active growth, the articular tissue of the condyle comprises firstly a

layer of dense fibrous connective tissue which forms the articular surface and is called the articular layer, secondly a subjacent layer of loose connective tissue termed the proliferative layer, because it contains proliferating pluripotent stem-cells, and thirdly a deep layer of hypertrophic hyaline cartilage (Figs. 37dd, 48v, 53n). While this basic appearance remains more or less constant throughout the period of growth, details change (H. Luder 1996). Cells of the articular layer, for instance, gradually assume a chondrocyte-like appearance around the time of puberty. Most major changes, however, occur during the first years after birth. Thus, condyles from newborns and infants regularly exhibit soft tissue columns containing large blood vessels, that protrude downwards from the articular layer, invaginate the proliferative layer, and extend through the hyaline cartilage to the subchondral marrow spaces (Fig. 19g). These columns which are also referred to as vascular canals and are considered remnants of the embryonic pattern of blood supply, normally disappear gradually during infancy. Also, during the first few years of postnatal life, the hypertrophic layer that is most prominent at birth, decreases dramatically in thickness. Subsequent age-dependent changes in the width of the hyaline cartilage, however, hardly exceed individual variation (Figs. 37dd, 48v, 53n, 79q).

The association between the various articular tissue layers and condylar growth is evident from experimental studies in suitable animal models that resemble humans with respect to the microscopic structure of the condyle (H. Luder 1996). The proliferative layer contributes to condylar growth by producing new cells committed to subsequent differentiation into chondrocytes, which are in apposition on top of the hypertrophic layer. Through this apposition of new cells, the hypertrophic cartilage grows in thickness and older cartilage cells are displaced deeper into the hypertrophic layer. As soon as new cartilage cells are fully differentiated, they start enlarging and synthesizing extracellular matrix. As a result, cells of the hypertrophic layer become separated from top to bottom by widening septa of the matrix and at the same time grow in size. Once chondrocytes have attained their final stage of hypertrophy at the lower border of the hypertrophic layer, most of them die, collapse, and leave empty lacunae for subsequent endo-

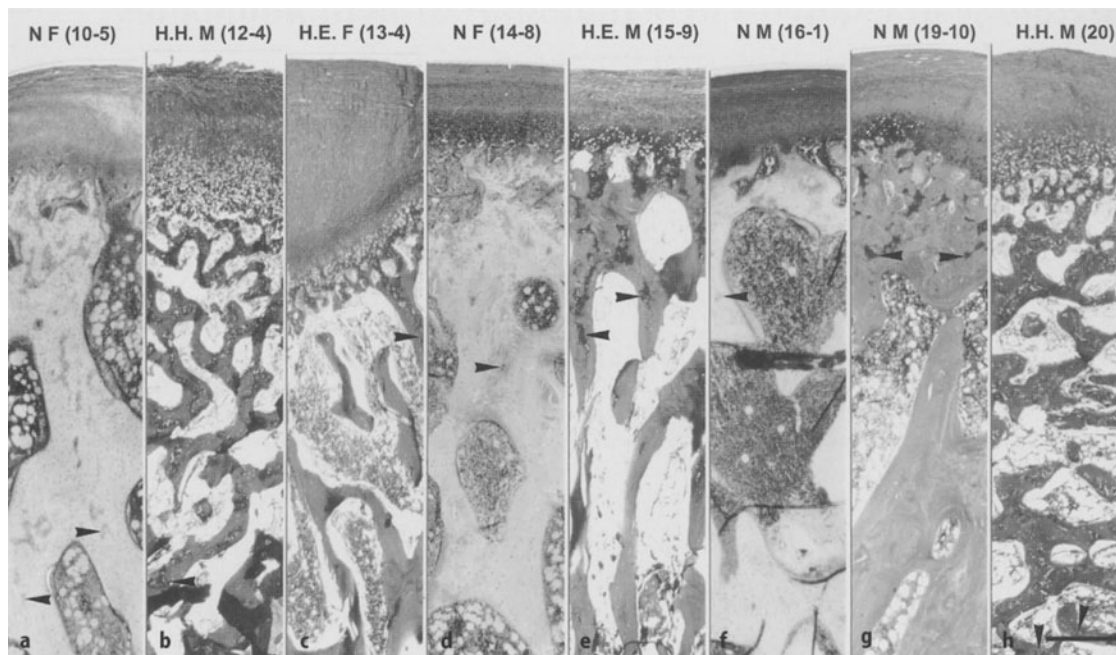


Fig. 79a–h. Histological findings in cases of condylar hyperactivity. Comparative age-dependent appearance of the articular tissue and subchondral bone in normal (N) condyles (**a, d, f, g**) as well as specimens from H.H. (**b, h**) and H.E. (**c, e**) cases at the growth stage (M male, F female, age in years-months). Note the cone-shaped thickening of the articular tissue in one of the H.E. cases (**c**) as well as the pattern of trabeculae and depth of cartilage islands (*arrowheads*) in the subchondral spongiosa. To appreciate the features of the spongiosa, note that the bone appears dark in the paraffin (**b, c, e, h**) and light in the plastic sections (**a, d, f, g**). Toluidine blue, original magnification $\times 33$ (*bar*=0.5 mm)

chondral ossification. This involves invasion by capillaries, chondroclasts which resorb cartilage matrix, and bone forming osteoblasts. Thus, endochondral ossification continuously converts into primary spongiosa what has been added to the hypertrophic layer through apposition of new cells, interstitial production of new matrix, and enlargement of the chondrocytes. In so doing, it maintains a more or less stable thickness of the hypertrophic layer.

During the conversion of the hyaline cartilage into primary spongiosa, chondroclasts do not resorb all of the extracellular matrix, but leave a cartilaginous scaffold for subsequent bone formation by osteoblasts. These primary trabeculae, which contain islands of cartilage, are remodelled only at some distance from the zone of erosion of the hypertrophic layer and replaced by trabeculae consisting only of lamellar bone. The depth of cartilage rests in the subchondral bone is apparently related to the rate of condylar growth (Fig. 79r). In specimens from newborns and infants, they can be traced as far down as the condylar neck. In individuals of more than 10 years of age, however, the distance of cartilage rests from the zone of erosion decreases dramatically, and in adults older than 30 years, they are confined to a very narrow subchondral band, if

present at all. This justifies the wide-spread practice of histologists of taking the frequency and depth of subchondral cartilage islands to decide, whether and at what rate growth had been active in specimens of condylar hyperplasia (M. Rushton 1944, 1946; T. Øberg et al. 1962; P. Egyedi 1969; J. de Burgh Norman and D. Painter 1980; L.G. de Bont et al. 1985; P. Slootweg and H. Müller 1986; H. Obwegeser and M. Makek 1986; F. Gray et al. 1990). However, it should be borne in mind that cartilage remnants are related primarily to cartilage erosion and could also occur when this process is slowed down or cannot keep pace with cartilage formation.

Considering the limited alterations of condylar microscopic appearance after infancy, which contrast strikingly with the variations observed in the rates of mandibular growth (A. Björk 1963), it would appear that a balance is normally maintained between cartilage growth and endochondral ossification, that keeps the thickness of the hypertrophic layer within narrow bands. Rather than variations in rates of growth, significant changes in cartilage thickness would thus reflect disturbance of the steady-state conditions between cell proliferation, matrix production, chondrocyte enlargement and cartilage erosion.

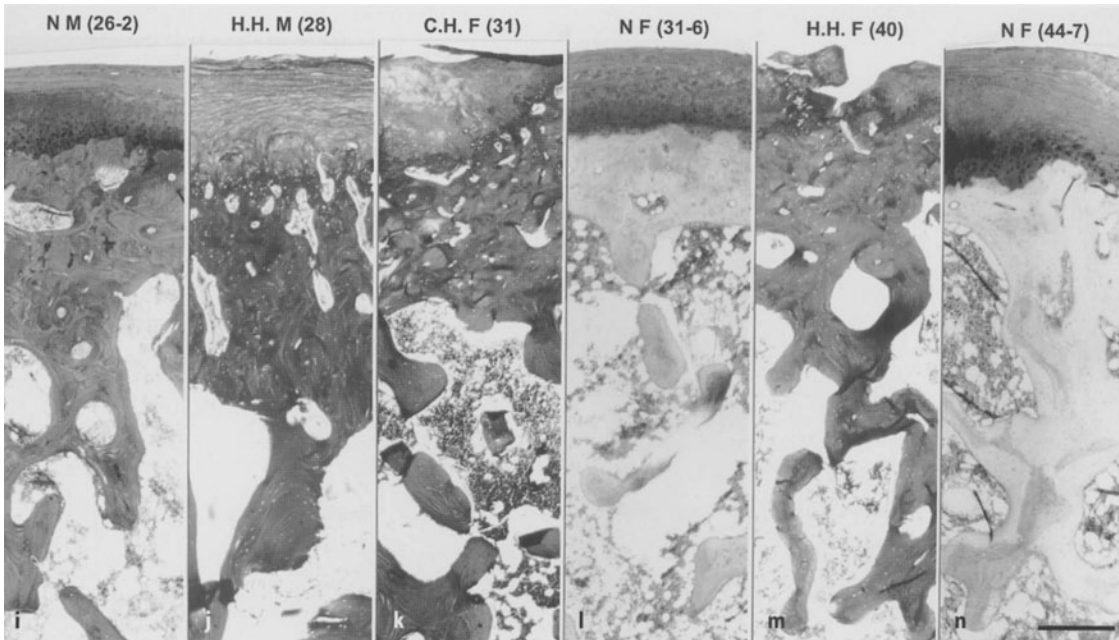


Fig. 79i-n. Histological findings in cases of condylar hyperactivity. Comparative age-dependent appearance of the articular tissue and subchondral bone in normal (N) condyles (i, l, n) as well as specimens from H.H. cases (j, m) and a case of "simple" condylar hyperplasia (C.H.; k) at the transitional and adult stages (M male, F female, age in years-months). Note the fraying of the articular surface in the C.H. case (k) and the vertical cleft across the entire articular tissue in the H.H. case (m). j, k, m paraffin sections, i, l, n plastic sections, toluidine blue, original magnification $\times 33$ (bar=0.5 mm)

Transitional Stage. The interval between late adolescence and the late twenties constitutes a period of transition from the microscopic appearance of the condyle during growth to the appearance prevailing for the rest of adulthood. The articular layer becomes almost entirely fibrocartilaginous and thereby is converted into the superficial zone of the adult articular tissue. In the proliferative layer, cell replication comes to a standstill, the density of constituent cells decreases, and some cells assume a chondrocyte-like structure. Unlike as claimed by some investigators (F. Gray et al. 1990), the layer does not disappear, although at this stage it should be more appropriately referred to as the intermediate zone. The hypertrophic layer is converted into the deep zone of the adult articular tissue. During this conversion, chondrocytes, although increasing in size from top to bottom of the zone, remain smaller than during growth, become separated by wider septa of matrix, and acquire prominent pericellular halos (Fig. 36l). In contrast to the situation during growth, the deepest calcified part of the cartilage becomes clearly visible as a distinct, darkly staining layer that is separated from the non-calcified tissue by a sharp line of demarcation, the so-called tide-mark (Fig. 39k). In the subchondral zone, a continuous bone plate is gradually built. Therefore, calcified cartilage borders in decreasing proportions on marrow spaces and in increasing proportions directly on bone

(Fig. 36l). In the marrow spaces, bone formation clearly predominates over cartilage resorption (H. Luder 1996). As a result, subchondral cartilage rests become scarce and located only in close proximity to the calcified cartilage (Fig. 79r).

Adult Stage. At the adult stage, the condylar articular tissue essentially constitutes fibrocartilage with varying prominence of fibrous and cartilaginous components (H. Luder 1997). In the superficial zone, chondrocytes occur scattered or in small groups, interspersed between masses of thick collagen fibres. In the intermediate zone, the proportion of chondrocytes rises further with age at the expense of fibroblasts, while the total number of cells continues to decrease. As a result, the remnants of the proliferative layer disappear as a distinct cell layer along an increasing proportion of the condylar articular surface. The fibrocartilage of the deep zone is characterized by scattered, small chondrocytes embedded in a lattice of obliquely arranged collagen fibres (Figs. 32n, 39k). Unlike the intermediate zone, the superficial and deep zones lack systematic age-related changes in appearance (H. Luder 1998). However, they can show signs of cartilage remodelling, mostly progressive remodelling characterized by the formation of chondrocyte clusters or the accumulation of cartilage matrix that is unusually rich in proteogly-

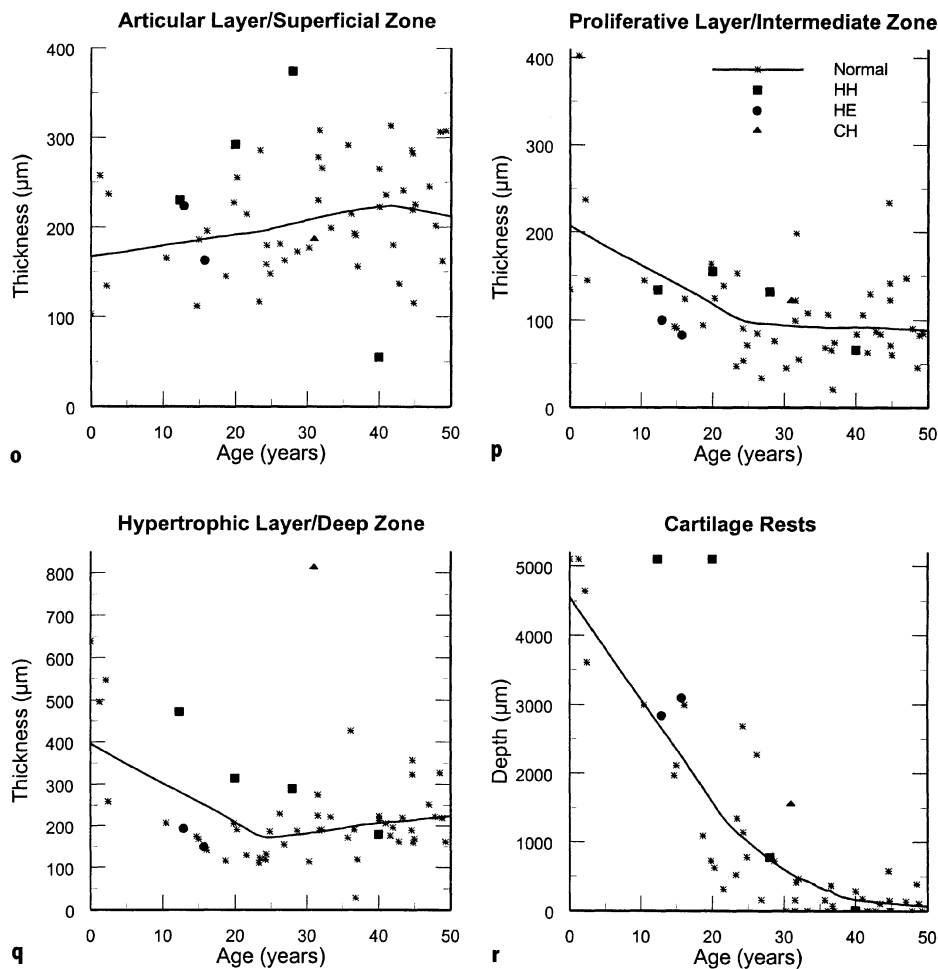


Fig. 79o–r. Histological findings in cases of condylar hyperactivity. Thickness of articular tissue layers or zone (o–q) and depth of subchondral cartilage rests (r) against age in normal condyles (asterisks and solid line indicating average) as well as specimens from H.H. (squares), H.E. (dots) and C.H. (triangle) cases. Please note that cartilage rests (r) in the H.H. cases of about 12.5 and 20 years of age, similar to those of normal newborns, were observed down to the resection border, which may not have coincided with the maximum depth

cans and, therefore, displays a strong metachromatic staining reaction with toluidine blue (Fig. 32n). This type of remodelling seems to be particularly frequent in individuals around 40–45 years of age and is associated with reactivated endochondral ossification that can produce small subchondral cartilage islands similar to those found during the period of transition from growth to adulthood (H. Luder 1998). In comparison with progressive remodelling, regressive remodelling involving dissolution of cartilage and its replacement by vascular fibrous tissue is very rarely seen in individuals below 50 years of age (H. Luder 1996). Similarly, signs of degeneration, i.e., osteoarthritis, are scarce in this age range. If present, degenerative changes are mostly confined to fibrillation of the articular surface and hardly

ever involve splitting or clefting of the deeper articular tissue zones (H. Luder 1996).

18.3 Condyles in Mandibular Growth Anomalies

18.3.1 Condylar Hypoactivity

The only specimen from this class of growth anomaly, that was available for histological examination, was the resected condyle of an 18-months-old girl with hemifacial microsomia. Not unexpectedly in such a case, this condyle was hypoplastic, its overall size corresponding approximately to that of a normal newborn. Interesting-

ly, however, the otherwise inconspicuous hypertrophic cartilage was also much thinner than normal, even when its thickness was related to the reduced overall size of the specimen. On the other hand, subchondral cartilage remnants were observed down to a depth of about 4.5–5 mm from the zone of erosion, which corresponds perfectly to the values found in normal age-matched specimens (Fig. 79r). Also, the trabecular pattern of the condylar spongiosa did not deviate significantly from that observed in controls (Fig. 19f,g). This could be taken to indicate that endochondral ossification and remodelling of the primary spongiosa in this condyle were fairly normal, whereas the production of hypertrophic cartilage through stem cell proliferation, matrix production and chondrocyte enlargement was deficient.

18.3.2 Condylar Hyperactivity

From cases of unilateral condylar hyperactivity, seven resected condyles were obtained as paraffin blocks embedded earlier for routine pathological diagnosis. Four of these cases, one female (Fig. 32) and three males (Figs. 36, 37, 68), were classified as hemimandibular hyperplasias (H.H.) or hybrid forms with a predominance of H.H., one case of a female (Fig. 39) as “simple” unilateral condylar hyperplasia (C.H.) and two cases, one female (Fig. 48) and one male (Fig. 53), as hemimandibular elongation (H.E.). Based on the patients’ ages at condylectomy, one specimen of H.H. and both specimens of H.E. had been removed during the growth period, two specimens of H.H. at the beginning and end of the transitional stage, respectively, and the last specimen of H.H. as well as the one condyle with C.H. at the adult stage. From all the existing paraffin blocks, new sections were cut, stained with toluidine blue for better comparison with control material, and analysed histometrically using a light microscope equipped with an eyepiece micrometer.

In addition, micrographs taken earlier from condyles of five patients were available for a qualitative examination. These cases were classified as H.H. at the growth stage (Fig. 31), a hybrid form with predominance of H.H. (Fig. 74), a hybrid form with predominance of H.E. (Fig. 73), H.E. (Fig. 28) and a hybrid form without predominance of either the H.H. or H.E. component (Fig. 69). A summary of the histopathology findings in all these specimens is displayed in Table 1.

18.3.3 Hemimandibular Hyperplasia

All condyles removed from cases of H.H. at the growth stage as well as one specimen from a male hybrid case with predominance of H.H. resected at the beginning of

the transitional stage exhibited regular hypertrophic growth cartilage and active or even intensive endochondral ossification. Thus, they constituted cases of condylar hyperplasia type I as defined by P. Slootweg and H. Müller (1986). Compared with normal condyles, the two specimens from males available for histometric analysis revealed some, although insignificant, thickening of the hypertrophic cartilage and abundant subchondral cartilage rests at significantly greater distance from the zone of erosion (Figs. 79q,r). The spongiosa comprised comparatively small marrow spaces interspersed in an abnormally tight, almost tumour-like network of thin, mixed cartilaginous-bony trabeculae lacking clear orientation (Figs. 79b,h). This pattern suggests that endochondral ossification and remodelling of the primary spongiosa cannot keep pace with cartilage growth, either because the latter processes are reduced or cartilage growth is increased in rate. Imbalanced or disturbed endochondral ossification and remodelling of the primary spongiosa could indeed explain the grossly distorted condylar shape that is frequently observed in cases of H.H.. How such a mechanism could account for the malformation of the mandibular ramus, angle and corpus, remains, however, mysterious. Rather than being responsible for these malformations, the abnormal endochondral ossification and remodelling of the condyle could also conceivably be the result of an as yet unknown, common cause that interferes with growth and remodelling of the entire hemimandible.

Apart from being compatible with the clinical picture, the trabecular pattern seen in the preceding first two cases also seemed to be typical of cases of H.H., thus allowing a distinction from cases of H.E.. Yet the histological appearance evident from micrographs of a condyle from a female patient classified as a “classical” case of “pure” H.H. (Fig. 31) did not fit the description given above. Rather, the condylar spongiosa gave an impression of normality, although the depth of subchondral cartilage remnants could not be ascertained. This suggests that the mechanisms leading to H.H. may vary, which would mean that a histopathological diagnosis based on condylar appearance alone would not be possible.

The only condyle removed during the transitional stage of development from a male patient suffering from H.H. revealed partly fibrocartilage, as typically seen in adult specimens, and partly a type of cartilage characterized by hypertrophic chondrocytes interspersed between arcades formed by prominent collagen fibre bundles. The latter type of cartilage resembled that described as occurring in condylar hyperplasia type II (P. Slootweg and H. Müller 1986). In agreement with this classification, there were signs of active endochondral ossification, which, however, did not exceed those seen in normal specimens (Fig. 36l), and a subchondral bone plate which exhibited abnormally numerous marrow

Table 1. Summary of histopathological findings in cases of H.H. and H.E. as well as hybrid forms and a case of “simple” condylar hyperplasia (C.H.)

Clinical diagnosis ^a	Case	Sex	Age ^b	Cartilage	Endochondral ossification	Subchondral marrow spaces	Subchondral bone trabeculae	Cartilage rests
H.H.	37	Male	12-4	Regular hypertrophic	Intensive	Narrow	Numerous, thin, ill-oriented	Numerous, deep
H.H.	31	Female	16-6	Regular hypertrophic	Active	Inconspicuous	Inconspicuous in number and size	Inconspicuous in number and depth
Hybrid, H.H. ≫ H.E.	68	Male	20	Regular hypertrophic	Intensive	Narrow	Numerous, thin, ill-oriented	Numerous, depth
H.H.	36	Male	28	Partly hypertrophic, partly fibrocartilage	Active	Slightly increased in number	Inconspicuous	Present
H.H.	32	Female	40	Fibrocartilage with severe degenerative changes	Missing	Inconspicuous	Inconspicuous	Absent
Hybrid, H.H. ≫ H.E.	74	Female	40	Fibrocartilage with severe degenerative changes	Missing	Inconspicuous	Inconspicuous	Absent
C.H.	39	Female	31	Fibrocartilage with initial degenerative changes	Minor	Inconspicuous	Inconspicuous	Few
H.E.	48	Female	13-4	Regular hypertrophic	Active	large	Few, slender, well oriented	Inconspicuous in number and depth
Hybrid H.E. ≫ H.H.	73	Male	13-10	Regular hypertrophic	Intensive	Large	Few, slender well oriented	Inconspicuous in number and depth
H.E.	53	Male	15-9	Regular hypertrophic	Active	Large	Few, slender, well oriented	Inconspicuous in number and depth
H.E.	28	Female	17-6	Transitional from hypertrophic to fibrocartilage	Minor	Large	Almost compact bone plate and slender trabeculae	Few
Hybrid	69	Male	16-10	Regular hypertrophic	Intensive	Inconspicuous	Inconspicuous	Numerous, deep

^a In hybrid forms, predominance of either the H.H. or H.E. component is indicated by “greater than”-signs (>)

^b Age (years-months) at condylectomy

spaces in direct contact with cartilage. This indicates regular, well-coordinated condylar growth continuing beyond the age when it normally terminates.

All condyles removed at the adult stage, i.e., two specimens from H.H. cases and the one from the only case of “simple” condylar hyperplasia, notably all females, revealed degenerative disintegration of the articular tissues, i.e., signs of osteoarthritis, but none of significant on-going growth. This characterization corresponds to that given by P. Slootweg and H. Müller (1986) regarding condylar hyperplasia type III. In the condyle from the youngest individual, degenerative changes were superficial and mild, whereas in those obtained at about 40 years of age they were severe, affected all zones down to the osteochondral junction, and were accompanied by marked regressive remodelling. These alterations clearly exceeded those found in age-matched control specimens and, therefore, cannot be considered “normal” ageing-associated changes. However, it cannot be decided, whether they presented the cause or effect of the abnormal condylar enlargement and distortion.

18.3.4 Hemimandibular Elongation

All condyles obtained from cases of H.E. or from hybrid forms with a predominance of the H.E. component were removed at the growth stage. All but one revealed the normal layered appearance of the articular tissue typical of this stage. The one exceptional specimen, shown already in a previous publication (H. Obwegeser and M. Makek 1986), exhibited a central cone-shaped thickening of the articular layer, that displaced the proliferative and hypertrophic layers downwards. This peculiar structure that, although lacking any blood vessels, resembled somewhat the vascular canals found in condyles from infants, could not be observed in any other specimen. Thus, unlike the assumption of H. Obwegeser and M. Makek (1986), it cannot be considered typical of H.E. cases.

In all condyles of this type of growth anomaly, including that with the local thickening of the articular layer, the hypertrophic cartilage exhibited a perfectly normal structure and thickness (Fig. 79q), and there

was obviously active endochondral ossification. According to the classification of P. Slootweg and H. Müller (1986), they all constituted condylar hyperplasia type I, similar to the specimens from H.H. cases removed at the same age range. However, there were some striking differences. In contrast to specimens removed because of H.H., those from H.E. cases disclosed subchondral cartilage rests down to normal distances from the zone of erosion (Fig. 79r), and the condylar spongiosa was characterized by slender, well-oriented, sometimes almost parallel bone trabeculae arranged between comparatively large marrow spaces (Fig. 79c,e). This suggests enhanced but well-coordinated cartilage growth, endochondral ossification and remodelling of the primary spongiosa, i.e., a growth pattern fitting well to the clinical and radiographic appearance of H.E..

18.3.5

Hybrid Forms

The only condyle from a case classified as hybrid form without predominance of either the H.H. or H.E. component (Fig. 68) was removed at the growth stage and had to be examined based on old micrographs alone. These micrographs indicated uniform thickening of the articular tissue, but did not allow identification of the proliferative layer. Regarding structure, the hypertrophic cartilage and the trabecular pattern of the subchondral spongiosa appeared inconspicuous. The conclusion that can be derived from these limited findings is that the histological appearance of the condyle, lacking any sign of either H.H. or H.E., fairly well reflected the clinical picture.

18.4

Summary

Irrespective of the clinical classification of the condylar hyperactivity cases, the histological appearance of excised condyles presented in this work is in good agreement with the descriptions given in the literature on condylar hyperplasia (M. Rushton 1944, 1946; L. Schultz et al. 1960; T. Øberg et al. 1962; R. Walker 1967; P. Egyedi 1969; J. de Burgh Norman and D. Painter 1980; L. de Bont et al. 1985; P. Slootweg and H. Müller 1986; H. Obwegeser and M. Makek 1986; R. Gray et al. 1990). These observations suggest that the microscopic structure of the cartilage covering the condylar bone only depends on the age at condylectomy, supporting the notion of R. Gray et al. (1990) that the types of condylar hyperplasia distinguished by P. Slootweg and H. Müller (1986) mainly reflected the different stages of physical development at onset of the growth disorder. In contrast to the report of R. Gray et al. (1990), however, the findings obtained

from the present investigation do not suggest that significant changes in the thickness of the articular tissue are associated with any of these growth anomalies. Rather, alterations at the cartilage level proved to be very subtle and did not exceed the range of normal variation. It would appear, therefore, that nature fairly successfully maintains a steady-state between the various growth components, even if overall growth of the condyle is highly disturbed. As a consequence, a distinction of the different clinical forms of growth anomalies based on the microscopic appearance of condylar cartilage does not seem possible.

The histological features that provided the most reliable, although not absolutely flawless, discrimination of H.H. and H.E. cases of the present material were the trabecular pattern of the condylar spongiosa and the depth of the cartilage remnants embedded in the subchondral bone trabeculae. While cartilage rests have consistently been considered by previous investigators (M. Rushton 1944; T. Øberg et al. 1962; P. Egyedi 1969; J. de Burgh Norman and D. Painter 1980; L. de Bont et al. 1985; P. Slootweg and H. Müller 1986; H. Obwegeser and M. Makek 1986; R. Gray et al. 1990), usually to decide whether or not condylar growth was active, the arrangement of trabeculae, to the best of my knowledge, has not been taken into account so far. The validity of this diagnostic feature, therefore, needs confirmation from further studies.

If proven to be useful, the appearance of the spongiosa would enable a distinction between normal, H.H.- and H.E.-condyles, as long as growth has not yet come to a standstill. Once growth ceases, however, subchondral bone trabeculae apparently remodel, thus assuming an inconspicuous arrangement, which no longer offers clues for a diagnosis. Moreover, even in cases operated on during active enlargement of the condyle, the characteristics of the spongiosa do not allow a decision to be made whether condylar growth was hyperactive or normal at the time of the surgical intervention.

Although virtually all previous reports on cases of condylar hyperplasia have speculated about its aetiology, this issue is still far from being clear. Considering the low numbers of uniform cases and the limited conclusions that can be derived from the minor alterations in the microscopic appearance of the condyles, histopathology is unlikely to offer useful explanations regarding aetiology and pathogenesis of mandibular growth anomalies. From the distinct histological manifestations of growth disturbances setting in during and after the normal growth period, it would appear that respective causes vary as well. Also, distinct aetiologies may account for H.H. and H.E., as the components of condylar growth in these two types of anomalies seem to be affected to strikingly different extents.

Principal Surgical Procedures

Principal Standard Operation Techniques and Instruments

The treatment of facial skeletal deformities due to mandibular growth anomalies cannot be limited to mandibular procedures only. The treatment plan and its execution must include all additional parts of the facial skeleton which are either directly malformed by the mandibular anomaly or which may exist independently of it but will have to be corrected simultaneously. Naturally, the mandibular sagittal splitting procedure, the Le Fort I-type mobilization, the chin and masseter hypertrophy corrections and tongue reduction techniques will be dealt with in more detail than the anomalies of the upper half of the facial skeleton. The possibilities for correcting the latter we owe solely to the ingenious inventions of P. Tessier (1967, 1968); P. Tessier et al. (1967). I want to include these possibilities using a very few cases only.

Frequently I have been asked about the historical background of the procedures which I had developed and which nowadays are used as standard procedures all over the world and how I conceived the ideas to produce them. It gives me much pleasure to recount what I remember about this. It has given me also deep satisfaction to research the literature in great detail since I had now, after my retirement, time enough to do so.

Ideas for new procedures do not fall from the clouds like raindrops. They are always the result of intellectual effort to find a solution to an existing problem. *"It is not difficult to find a solution to a problem, it is only difficult to identify the problem"* (Principle No. 36). New good ideas are the consequence of being repeatedly confronted with the same problem. And they are born on the basis of experience and knowledge. Once one has recognized a certain problem and believes a solution to that problem has been found, it does not mean at all that that solution is the ultimate answer to that problem. That solution may be the basis for further improvement and an even better answer.

In surgical techniques the ultimate best procedure may finally be produced by someone else who has realized that an additional improvement is needed and possible. Or it is the originator himself who has improved his own techniques, maybe through new and better instrumentation, through observed complications with the original procedure or through recognition of additional needs in a variety of cases. Once a procedure has

become a standard operation technique, the possibility of additional variations or alterations is not excluded. But it will still always remain the originator's primary idea and basic procedure.

It is almost unbelievable how often colleagues publish such procedures as if they were themselves the originators. I also do not understand why, so often in publications, the drawings of an original technique of the author are reproduced as if this were his present day method, although he has improved his technique and published it many times. This indicates reprehensible inefficiency in searching the current literature. A typical sample is the sagittal splitting procedure on the ascending rami. It is often reproduced in publications (the drawings taken mostly from my papers and articles published in 1955 and 1957) as if this was my present-day technique. However, my current methods for the rami sagittal splitting procedure are not one but several variations depending on the requirements of the case. I have published these variations many times, including the reasons for using them.

A second reason besides the historical background, why I want to mention these procedures is that rather often through questions of colleagues, I gather that they are having occasional problems with the standard operation techniques for the correction of maxillo-mandibular disharmonies. Frequently, questions arise about the incidence of damage to the mandibular canal contents and other problems with the sagittal splitting procedure. There is again and again the question of how to mobilize the maxilla properly and to avoid relapse after advancing it, particularly in cleft cases. Some colleagues seem to have problems with the transoral chin correction and even several very experienced surgeons have asked me, not too long ago, about which techniques I use for the correction of masseter hypertrophy and tongue reduction. For this reason, I will discuss these procedures and how I did these standard operations using my latest methods. I will also discuss the main possible complications during and after surgery, how to deal with them and how to avoid them.

When I developed these procedures, I had to use ordinary surgical instruments. When certain surgical problems arose repeatedly I had special instruments developed in order to overcome these difficulties, in par-

ticular to avoid complications during and after surgery. These instruments have come on the market under my name. It grieves me what poor quality instruments are often sold as “Obwegeser Instruments”. Some of them are very poor copies of my “Original Obwegeser” instruments. Others are just old patterns of my original instruments. With instruments it is the same as with surgical techniques: through experience, one constantly improves both the operation techniques as well as the instruments.

For that reason I will show how the more important newer ones are applied in each specific operation technique. I will also give information on which companies are producing my instruments and permit me to check them for correct design and quality and therefore have the right to call my instruments “Original Obwegeser” and put my name on them.

The Sagittal Splitting of the Mandible Procedure

20.1

Historical Background

I have often been asked how I conceived the idea of the sagittal splitting procedure on the mandibular ascending rami. The story is as follows: I started my training in maxillofacial surgery in 1947 with my teacher Richard Trauner at the Maxillofacial Unit of the Dental School of the University of Graz, Austria. At that time orthognathic surgery was almost non-existent with the exception of the occasional correction of a then so-called prognathism case. The Kostečka procedure was used, with the patient sitting in a dental chair. Before surgery he had received some sedation, premedication then local infiltration and block anaesthesia. An assistant held the patient's head steady. With the help of a large curved awl, a strong thread was pulled around the lingual side of the mandible, above the lingula. To that thread a Gigli saw was fixed and pulled through the soft tissues and the covering skin. With that Gigli saw the ascending ramus was cut. It took just 15 min or less to cut both sides. Then the jaw was immobilized by intermaxillary fixation for 6–8 weeks in the preplanned and prepared occlusion.

In 1952, my chief, Professor T. Trauner wanted me to follow up all of our 36 cases of Kostečka operations performed for correction of prognathism. The late results were not any better than those reported by other investigators. Roughly about 50% of the cases had some remaining unfavourable complications: partial or total relapse, open bite, pseudarthrosis, irreversible damage to the mandibular nerve and even worse to the facial nerve, parotid gland fistula, etc.. The occlusal relapses, Trauner assumed, were due to inadequate bony union because of the too-small contacting bone surfaces of the two fragments, partially due to the horizontal osteotomy above the lingula, where the ramus is very thin and additionally due to the anterior upwards rotation of the proximal fragment caused by the pull of the temporalis muscle. Because of this, he sought a procedure which would secure better bony union through more broadly contacting bone surfaces. He asked me to think about it and stated he would also do so himself.

I found all formerly published procedures from the literature. K.E. Hogemann (1951) had mentioned all of

them in his famous publication on “Surgical Orthopaedic Correction of Mandibular Protrusion”. I did not like all the ones with an extraoral approach because of the unavoidable scar in a visible area of the face. The very few transoral techniques did not produce the required long contacting bone surfaces. In order to solve this problem I asked myself “What is my goal?”: I wanted an osteotomy procedure which could be performed transorally only, avoiding any skin incision. It should also produce a large contacting bone area and avoiding damage to the contents of the mandibular canal. With these three important considerations in mind, I held a cadaver mandible in my hands, turned it around several times and got the idea of the sagittal splitting of the ramus via an oral approach.

In those days I had seen and treated conservatively a great number of jaw fractures. By experience, I had learned that we hardly encountered any infection in the fracture line, even without antibiotics, when we could immobilize the fragments within 24 h, even when they were not properly repositioned by elastic traction. Transoral surgical repositioning of mandibular fractures and fixation with screws and plates were not then in use. On radiographs I had twice seen a fracture almost equivalent to a sagittal splitting of the ramus. With this in mind, I imagined that I should be able to produce such a sagittal fracture of the ramus surgically.

In order to find out how much cancellous bone exists between the outer and inner cortical plates and where the mandibular nerve is located, I cut a dry ascending ramus horizontally at various levels, from the angle up to the semilunar notch. It became obvious that a sagittal splitting of the ramus should not be too difficult if the inner and outer cortical plates were cut first at different levels and these two cuts connected. Then I tried to perform a sagittal splitting of the ascending ramus at the Institute of Anatomy. It worked to my satisfaction. I showed it to my teacher R. Trauner and told him that we should be able to perform it transorally. I always hated skin incisions for surgery meant to improve the patient's appearance. Trauner accepted the idea. He said that Perthes and Schlössmann had already tried sagittal splitting of the rami from an extraoral approach.

Since that time I had believed that Perthes and Schlössmann were the first to have the idea of a sagittal

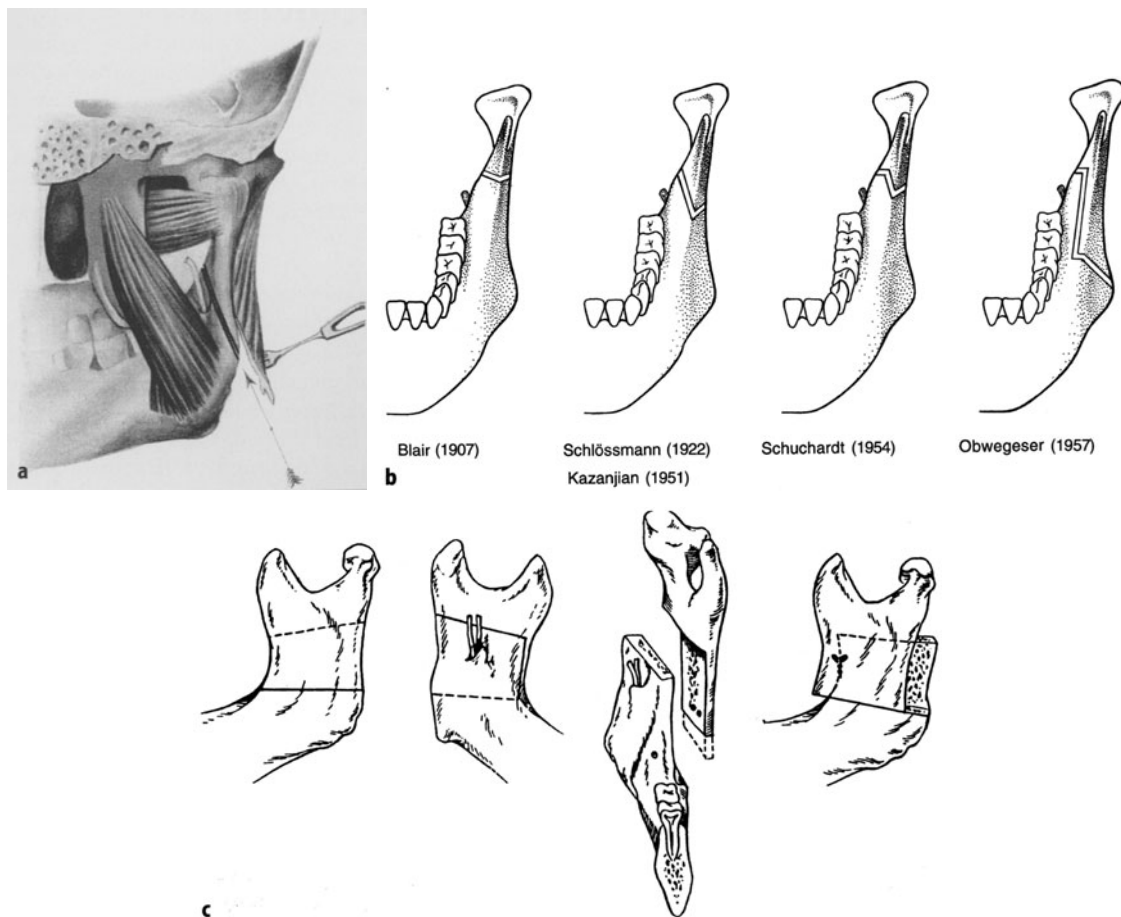


Fig. 80a–c. Steps in the development of increasing the amount of contacting raw bone surfaces in osteotomies of the ascending ramus. **a** Schlössmann's oblique osteotomy of the ascending ramus (from Perthes 1924). **b** Historical background of osteotomies at the ascending ramus in the sagittal plane. **c** Drawing of the first sagittal splitting procedure on the ramus (from R. Trauner and H. Obwegeser 1955)

splitting procedure on the ramus for corrective surgery of mandibular anomalies. In my retirement I have now had time to search for their original publication of 1924. I found it published by G. Perthes only but I also found a publication by G. Perthes (1922) in which he mentions that for the correction of a prognathic mandible he has used the procedure of A. Schlössmann (Fig. 80a). So, there is no Perthes-Schlössmann procedure. It is the procedure obviously first done by A. Schlössmann and then also used and published by G. Perthes (1924) as the Perthes-Schlössmann procedure. He wrote "We osteotomize the ascending ramus close to its transition into the body in an oblique plane from lateral below to medial above". From a submandibular incision they first detached the tuberosity of the angle with the insertion of the masseter muscle, using an osteotome to chisel it off. Next, with a drill bur, several bur channels were made from lateral below to medial above, starting at the occlusal level of the lower molars. The ramus was then cut

along these bur channels with a sharp osteotome. That is the description of the procedure in the original publication. So it was not a sagittal splitting procedure of the rami at all. It was the desire for longer contacting osteotomy surfaces than was the horizontal osteotomy above the lingula.

V. Blair's (1907) horizontal osteotomy produced very small contacting bone surfaces and included the disadvantage that the pull of the temporalis muscle easily rotated the proximal fragment upwards. In order to prevent that rotation F. Skaloud (1951) had suggested pulling a wire through the ramus just inferior to the osteotomy line and bringing it over the semilunar notch to the buccal side where it could be twisted together with the other end. A. Schlössmann's (1922) and V. Kazanjian's (1951) oblique osteotomy produced broader contacting bone surfaces. However, they did not become easily applicable and popular procedures.

K. Schuchardt (1954), after he had assisted me with a sagittal splitting procedure on the rami, had then suggested also an oral approach for performing an oblique osteotomy on the ramus which, in his operation picture, turned out to be more step-like than oblique as he had described it. It was finally the sagittal splitting procedure which produced really long contacting bone surfaces (Fig. 80b). But it had to be proven first.

20.2

My First Two Cases of the Sagittal Splitting Procedure

20.2.1

The First Attempt

The first case to be presented was a 27-year-old female (Fig. 81a,b). She was edentulous and quite prognathic. Acrylic splints were prepared in the desired intermaxil-

lary relationship. Professor R. Trauner decided that we would operate together on the first case. On one side he would perform a vertical osteotomy of the ascending ramus from a sub-mandibular approach. On the other side, my idea of splitting the ramus sagittally should be tried.

With the sagittal splitting procedure of the ramus, the greatest area of contacting raw bone surface of all the possible osteotomies of the ascending ramus was produced (Fig. 80c).

UNIVERSITÄTSKLINIK für Zahnheilkunde und Kieferstation im Landeskrankenhaus Graz		Stationär 3485/53
		Ambulant
1. Aufnahme <u>10.2.53</u>	19	Journal-Nr. <u>5375/53</u>
2. Aufnahme <u>16.4.53/14323/53</u>	19	Photo <u>10.2.53/15.4.1953</u>
3. Aufnahme	19	Medell <u>9.5.53</u>
transferiert von		Amb.-Pr.-Nr.
Vor- und Zuname: <u>Gersdorfer Johanna</u>		
Alter und Beruf: <u>26.7.1926</u>		
Geburtsort und -land: <u>Zellberg</u>		
Adresse des Patienten oder der Angehörigen: <u>Mühlberg, Harz, 31</u>		
Diagnose: <u>schwere Prognathie bei zahnlosem UK</u>		
Schema:		
Therapie (Operation): <u>Progenie-Operation, Kinnabmeisslung, Alveolarkamm</u>		
<u>plastik</u>		
Datum der Operation: <u>17.2.1953</u>		
Operator: <u>Prof. Dr. Trauner</u>		
Narkose: <u>16.4.53, 25.4.53</u>		
Ass. Dr. <u>Obwegeser</u>		
Wundverlauf: lokal: allgemein:		
Histolog. (bakter.) Befund:		
Röntgenbefund:		
Sektionsbefund:		
Eintrittsgewicht: Austrittsgewicht:		
Bemerkungen:		
Geheilt, gebessert, ungeheilt, gestorben, transferiert auf		
1. Abgang	2. Abgang	3. Abgang
<u>7.3.1953</u>	<u>18.6.53</u>	
Verfasser der Krankengeschichte:		Revidierender Assistent:

Fig. 81a. First attempt at sagittal splitting of the ascending ramus (17 February 1953). Patient's record, front page

sor, assisting both of us. The patient was in the half-sitting position on the operating table. After premedication and block and local infiltration anaesthesia, first Professor Trauner did a vertical osteotomy from a skin incision in the right angle area using a hand-held keyhole saw. In the area where the fragments would overlap after repositioning the mandible, the cortical plates were trimmed using a large bur. The surgical wound was packed while the operation on the other side was performed.

Next, it was my turn to try to perform the first sagittal splitting of the ramus. Again under block and local infiltration anaesthesia the operation was performed with the patient in the half-sitting position on the operating table: the patient was asked to open her mouth wide. Then, the mucosa was incised along the left ascending ramus and the ramus freed by elevation of the periosteum on its lateral aspect, from in front of the angle area up to the semilunar notch and all the way back to the posterior border. The same was done on the lingual side above the lingula. I cut the lateral cortical plate, again using a keyhole saw, from the inner angle to the outer angle of the jaw. To cut the cortical plate above the lingula all the way back to the posterior border, I al-

so used the keyhole saw. Both cuts were connected along the anterior border of the ramus with a fissure bur. Next, I tried to split the ramus using an osteotome. The ramus shattered rather than split!

Then the splints were fixed to the mandible by circummandibular wires and to the maxilla by peralveolar wires. The mandible was set back and fixed in the planned position by wiring both splints together. A direct wire was placed on each side by the respective surgeon, plus a circummandibular wire on the side of the vertical osteotomy (Fig. 81c).

Some penicillin powder was scattered into the wounds. Then they were closed. The patient was given penicillin postoperatively. The postoperative course was uneventful. The intermaxillary wire fixation remained for 2½ weeks. Then the wires were replaced by elastics.

The first attempt at performing a sagittal splitting of the ramus was a poor performance, a mess rather than a sagittal splitting. It was not nearly as nice as I had hoped it would be. The technique still had to be improved, particularly through better instrumentation. However, finally the patient had the correction of her prognathism as we had planned (Fig. 81d,e) (R. Trauner and H. Obwegeser 1955).

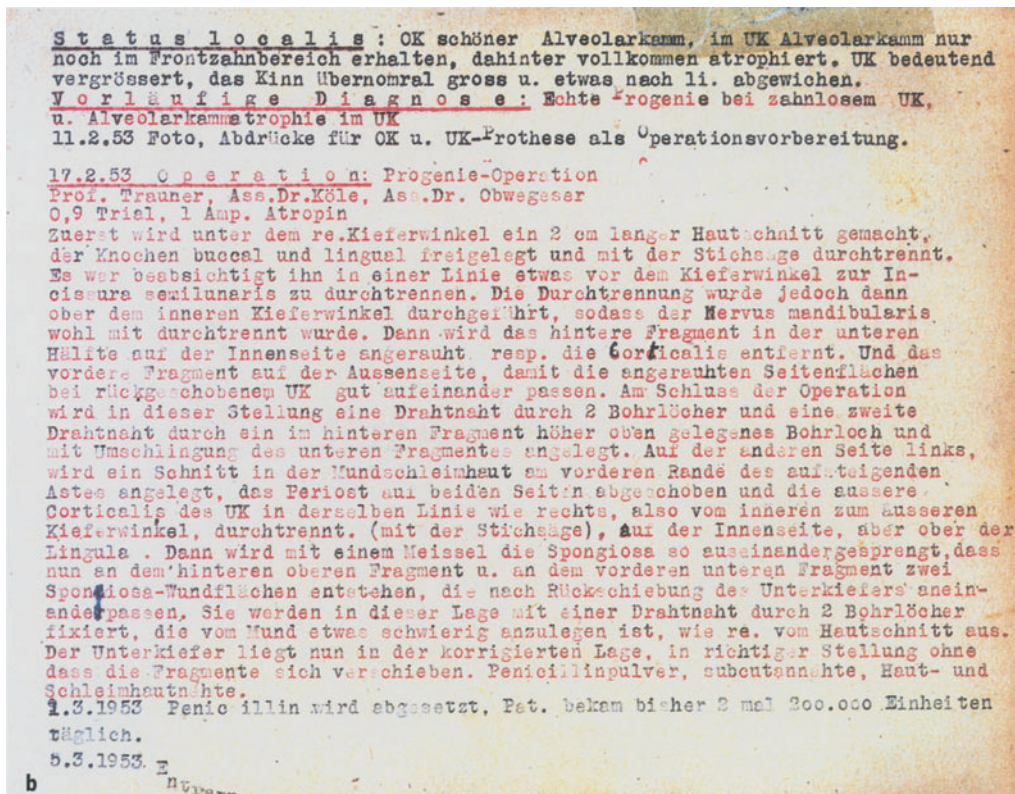


Fig. 81b. First attempt at sagittal splitting of the ascending ramus; operation report

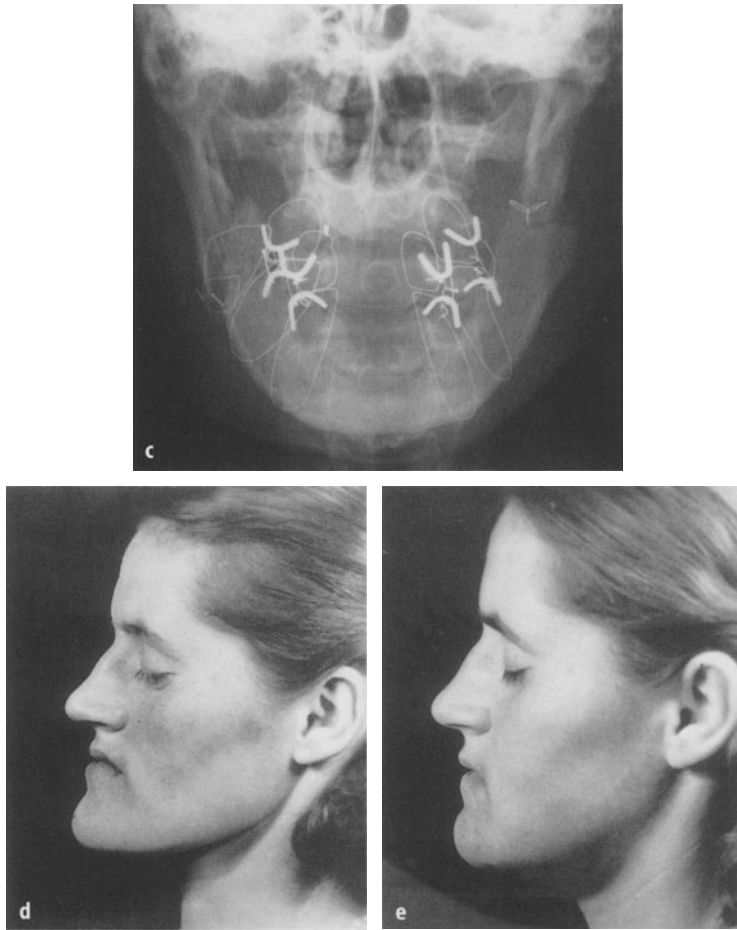


Fig. 81c-e. First attempt at sagittal splitting of the ascending ramus (17 February 1953). **c** P.a. radiograph of the mandible with intermaxillary fixation after vertical osteotomy on the right side by R. Trauner and sagittal splitting of the ramus on the left side by H. Obwegeser, on an edentulous patient. **d, e** Patient's profile before and 3 months after surgery

20.2.2 The Real-First Sagittal Splitting Procedure on the Ascending Ramus

The second case (Fig. 82a,b) arrived 2 months later, a 24-year-old lady quite prognathic. In the meantime Trauner had conceived the idea of the inverted L-shaped osteotomy of the ramus. He wanted to perform the vertical osteotomy cut behind the lingula from a submandibular approach and the horizontal cut above the lingula transorally.

For the correction of this prognathic mandible, it was intended that R. Trauner would perform his new idea of the inverted L-shaped osteotomy of the ramus on one side and on the other side, my idea of the sagittal splitting procedure (Fig. 80b) would be executed by myself.

The patient was a healthy young lady with a full dentition (Fig. 82c-e). The planning and the preparation of the case was done as was usual in those days: im-

pressions of the teeth were taken, the plaster models made and then an attempt was made to ascertain what the occlusion might be if the mandible was mobilized, repositioned and fixed to the upper teeth. Grinding of some teeth was necessary in order to achieve good interdigitation of the occlusion. Radiographs were taken in order to exclude any pathology within the mandible. After the desired grinding had been done, continuous loop wiring was applied to the upper and lower teeth (H. Obwegeser 1952) as a prerequisite for fixing the mobilized mandible to the upper teeth in the planned occlusion.

The operation was scheduled for 22 April 1953; again R. Trauner and H. Obwegeser were the surgeons, assisted by Prof. Schuchardt from Hamburg, who was visiting for 1 week. The patient was premedicated for sedation and again placed in a half-sitting position on the operating table. Again mandibular block and local infiltration anaesthesia were used.

UNIVERSITÄTSKLINIK für Zahnheilkunde und Kieferstation 22 a im Landeskrankenhaus Graz		Stationär <u>3562/53</u> Ambulant
1. Aufnahme	<u>20.4.53</u>	19
2. Aufnahme		19
3. Aufnahme		19
Transferiert von		Amb.-Pr.-Nr.
Vor- und Zuname: <u>Senekowitsch Mathilde</u>		
Alter und Beruf: <u>25.2.1929 Holzerbeiterin</u>		
Geburtsort und -land: <u>Graben, Jugoslawien</u>		
Adresse des Patienten oder der Angehörigen: <u>Lebern 27, Gem. Feldkirchen</u>		
Diagnose: <u>Progenie</u>		
Schema:		
Therapie (Operation): <u>Progenieoperation nach Trauner</u>		
Datum der Operation: <u>22.4.53</u>		
Operateur: <u>Prof. Trauner, Ass. Obwegeser</u>		
Wundverlauf: lokal: _____ allgemein: _____		
Histolog. (bakter.) Befund: _____		
Röntgenbefund: _____		
Sektionsbefund: _____		
Eintrittsgewicht: _____ Austrittsgewicht: _____		
Bemerkungen: _____		
Geheilt, gebessert, ungeheilt, gestorben, transferiert auf		
1. Abgang	2. Abgang	3. Abgang
<u>11.5.53</u>		
Verfasser der Krankengeschichte: <u>K. Senekowitsch</u>		Revidierender Assistent: <u>H. Obwegeser</u>

Fig. 82a. The real first sagittal splitting procedure on the ascending ramus (22nd April 1953). Patient's record, front page

Professor Trauner wanted me to operate first. The patient was asked to open her mouth. A type of mouth gag was inserted on the right side. On the left side, the mucosa was incised along the anterior border of the ramus, continued anteriorly in the vestibulum to the first molar region. The periosteum with the soft tissues was elevated first in the retromolar area and then on the whole buccal side and above the lingula on the inner side. The cortical cuts were attempted using Lindemann burs. One after the other broke. Then with the keyhole-saw, the lingual cut was made between the semilunar notch and the lingula. The buccal cortical cut just above the angle was also started with the keyhole-saw and then completed with fissure burs. These burs were also used to connect the two cortical cuts along the anterior border of the ramus. With the first blow with the osteotome, the coronoid process fractured off. All cortical cuts were now further deepened by the use of fissure burs. After that it became possible to split the ramus as desired with no further problems, to my astonishment and great satisfaction. Care was taken not to push the osteotome too deep in between the two cortical plates. By twisting the osteotome, the cortical plates opened gently and fi-

nally fell apart. As the other side was not yet mobilized we could not fix the fragments together by an anterior border wire. As the fragments were almost self-adapting, while moving the mandible back on the split side we decided wire fixation of the fragments might not be necessary if we closed the soft tissues including the periosteum properly. After we had spread some penicillin powder into the wound, we closed the soft tissues rather tightly and moved over to the other side.

The right side was operated on by R. Trauner in the way he had planned, as an inverted L-osteotomy of the ramus: the ramus was freed from an extraoral approach. Then an incision was made in the mucosa along the anterior ramal border. The horizontal bone cut was made below the semilunar notch with fissure burs. Then the vertical osteotomy behind the lingula was performed, also using fissure burs, via the extraoral-angle approach. I remember this very well, although it is not mentioned in the operation report. The mandible was repositioned into the planned occlusion and fixed to the upper teeth. On the anterior segment the lateral cortical plate was partially trimmed off with a burr in the overlapping region. Then a circumferential wire was used to

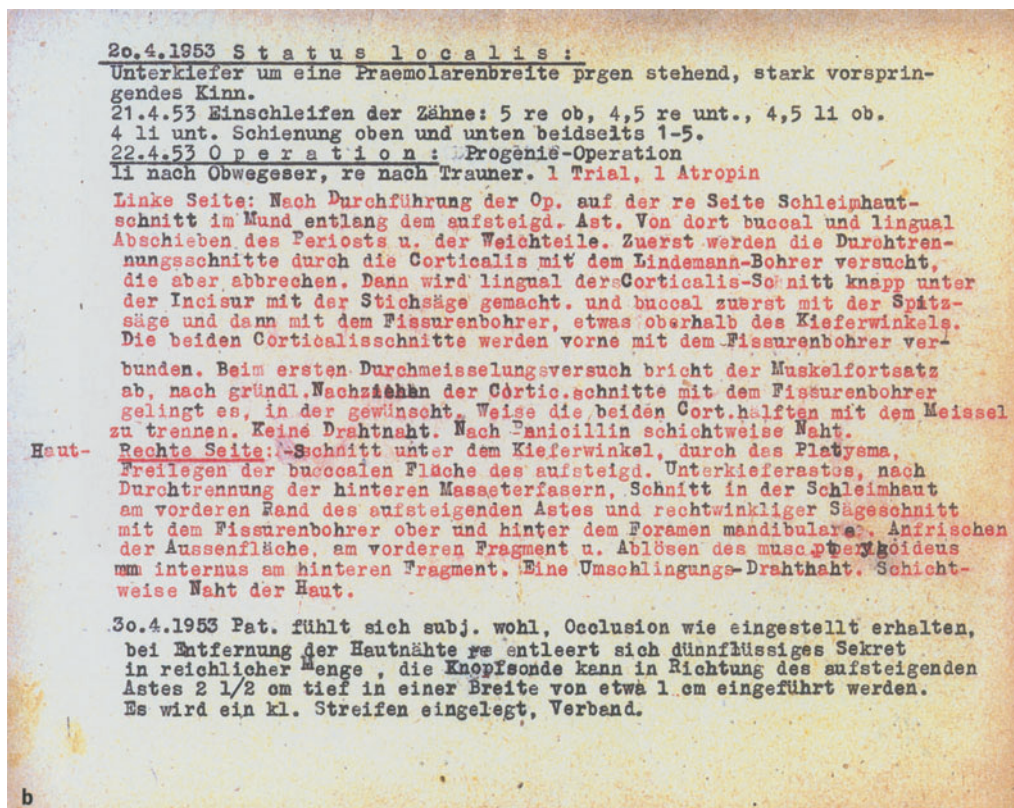


Fig. 82b. The real first sagittal splitting procedure on the ascending ramus (22nd April 1953). Patient's record, operation report

hold the overlapping fragments together followed by 6 weeks intermaxillary fixation (Fig. 82f). The postoperative course was uneventful. At follow-up 5 months after surgery, the patient had a satisfactory appearance and occlusion (Fig. 82g–j). There was nothing unpleasant visible on the left side, where the sagittal splitting procedure had been performed. The angle area looked clearly improved. In the right angle region, the scar resulting

from the submandibular incision was rather wide and reddish. There was anaesthesia of the lip on the side of the sagittal splitting which disappeared completely within 1 year. The appearance and the improved angle area and the occlusion remained unchanged, even after 33 years when my nephew J. Obwegeser called the lady in for a check-up (Fig. 82k,l). I consider this case to be my first real sagittal splitting of the ramus.

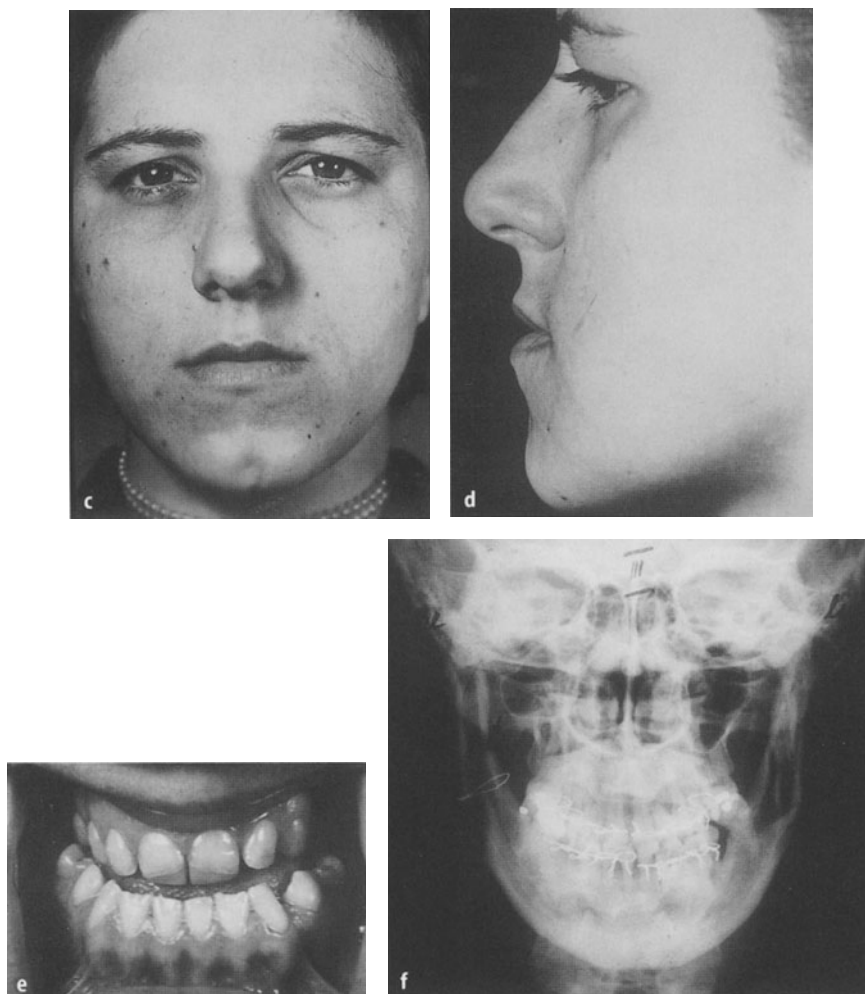


Fig. 82c–f. The real first sagittal splitting procedure of the ascending ramus (22nd April 1953). **c–e** Patient's appearance and occlusion before surgery. **f** Mandible p.a. projection with intermaxillary fixation 4 weeks after inverted L-shaped osteotomy on the right side by R. Trauner and sagittal splitting of the left ascending ramus by H. Obwegeser, without wire fixation



Fig. 82g–l. The real first sagittal splitting procedure of the ascending ramus (22nd April 1953). **g–j** Patient's appearance and occlusion 1 year and 5 months after surgery. **k, l** Patient's appearance 33 years after my first real sagittal splitting procedure

20.3 The First Sagittal Splitting Procedure Under General Anaesthesia

This was performed on 9 April 1956. From 1953 until then, I did all cases under local and block infiltration anaesthesia with some additional sedation, in a half-sitting position. There were about five cases altogether. The first case performed under general anaesthesia I did in Zürich.

The chief of Orthodontics at the Zürich Dental School, Professor R. Hotz, and the chief of Oral-Maxillo-facial Surgery, Professor P. Schmuziger, had visited Graz on the occasion of the annual meeting of the Austrian Dental Association in 1952. My teacher R. Trauner, the president of that meeting, had organized a week of post-conference demonstrations. We demonstrated live oper-

ations in preprosthetic surgery, primary cleft lip and palate repair, tumour surgery, etc.. We showed the full scope of our work with patient follow-up. When these two guests from Switzerland watched me doing a sub-mucous vestibuloplasty on the maxilla and on another case, a lowering of floor of mouth procedure, I had good luck and they were impressed with my operations. The surgery was almost bloodless and was performed under local anaesthesia only. They talked to each other in the Swiss-German dialect which is not comprehensible unless one has lived there. I had been born and brought up in Western Austria, a state called Vorarlberg, which borders on Switzerland. Our dialect is almost the same

as their's. I understood that R. Hotz said to P. Schmuziger "This young man might be a good candidate for your succession". He intended to retire within a few years and there was no well-trained Swiss candidate available then. So they talked about such a possibility with my teacher R. Trauner. He suggested I should go to Switzerland for a trial year, beginning 1 August 1954. He wanted me to come back to his department again if there was no possibility of getting a number of our own beds in Zürich for our patients within a year. By the end of that trial year we had five beds. As we did not have our own operating rooms I was kindly accepted as a guest surgeon at the Department for General Surgery at the University Hospital, for 7 years. That was how the Zürich School of Maxillofacial Surgery started.

It happened that R. Hotz had a private patient from another European country. He did not want his colleague P. Schmuziger to operate on the case as he had experienced too many severe complications with the procedure P. Schmuziger was using. He also did not want to send the patient to France or Germany as he had formerly done, as he felt ashamed that in Switzerland such a case could not be operated upon. So he wanted me to do the case using the sagittal splitting of the rami procedure which had been published a year previously (R. Trauner and H. Obwegeser 1955). I was not at all enthusiastic as I had not performed the procedure for almost 2 years, since working in Zürich.

The patient was a very charming, very slim young girl. She was 14 years and 6 months old. Although she was at an age where a relapse could be expected, she had not shown any further growth or change in position of the mandible in the previous 6 months. She had a rather long and narrow mandible. Whenever possible she camouflaged her prognathic appearance by keeping the mandible in a slightly open position (Fig. 83a). In the closed position she had a clear antemandibulism intermaxillary relationship (Fig. 83b). She suffered from partial anodontia, of the mandible more than of the maxilla. All her teeth were rather small and all the lowers were tilted lingually. The lateral cephalogram disclosed that the maxilla was small and retrodisplaced (Fig. 83c). Her general health was unremarkable.

In those days there was no panoramic view. The p.a. projection of the mandible proved that her mandible was generally rather slim, both the horizontal as well as the ascending rami (Fig. 83d). I was curious to find out how the sagittal splitting would work on such narrow rami. As seen on the lateral cephalogram there was also a clear retromaxillism. At that time there was no chance of repositioning the maxilla forward. The only plan possible was to reposition the mandible so much posteriorly that postoperatively crown and bridgework could provide the girl with good chewing function.

The teeth did not look to be suitable for a wire splint for the necessary intermaxillary fixation. I had a cast

cap splint made for each jaw. They were cemented in place preoperatively.

Since the girl was a private patient from a foreign country, R. Hotz wanted his patient to be hospitalized in a nice private hospital and operated upon under general anaesthesia. The operation was scheduled for 9 April 1956. I had never operated before in that private hospital nor had I performed the procedure under general anaesthesia. I had to convince the anaesthesiologist that I would need naso-tracheal intubation. This was something new to the anaesthesiologist as well as to me. He was not greatly enamoured with that idea. The tube extended out of the nose quite a lot. I did not like this. I explained to him what I was going to do. He did not approve of my turning the patient's head from one side to the other. My chief was assisting me, together with a young doctor. Today's special instruments did not exist at that time. Indeed, I expected some difficulties.

First I secured the lower cap splint by placing two circummandibular wires in the anterior region. Then I started with the actual operation. I injected a vasoconstrictor solution into the field of the soft tissue and bone surgery. I started with the splitting of the right ramus.

As I was accustomed to freeing the lateral aspect of the ramus all the way up close to the semilunar notch and the same on the medial side above the lingula, I did that in this case also. I did the cortical cuts with Lindemann burs very carefully, no deeper than until some bleeding points were visible, indicating that I was approaching the cancellous bone. First I did the cut above the lingula, then the lateral cut from the inner to just above the outer angle. Then I connected both along the anterior rim, just lingual to it, by first making bur holes with a rosehead bur and then connecting these holes with a short Lindemann bur. I had learned the advantage of these bur holes having discovered that the fissure or the Lindemann bur easily slips across the rim laterally, almost facilitating a fracture of the ramus when twisting the wide thin osteotome for the splitting.

On the first side the splitting went smoothly, but under poor vision. It took me rather a long time. On the second side I ran into problems not previously experienced. The cortical cuts were done as on the first side. I did the splitting again by striking the thin, broad osteotome 5 mm deep only, and then twisting it. When doing so the lateral ramus part broke off as a separate fragment. As I had detached the periosteum on the whole lateral aspect of the ramus it was a free fragment which I could have taken out but did not dare to do so because of the watching professors. That was a bit of a shock to me as I had not experienced this before. This was my first sagittal splitting at my new position in Zürich and with two professors watching! Not a good advertisement for my procedure, in particular since my chief had told the students that this procedure could probably be done only on paper.

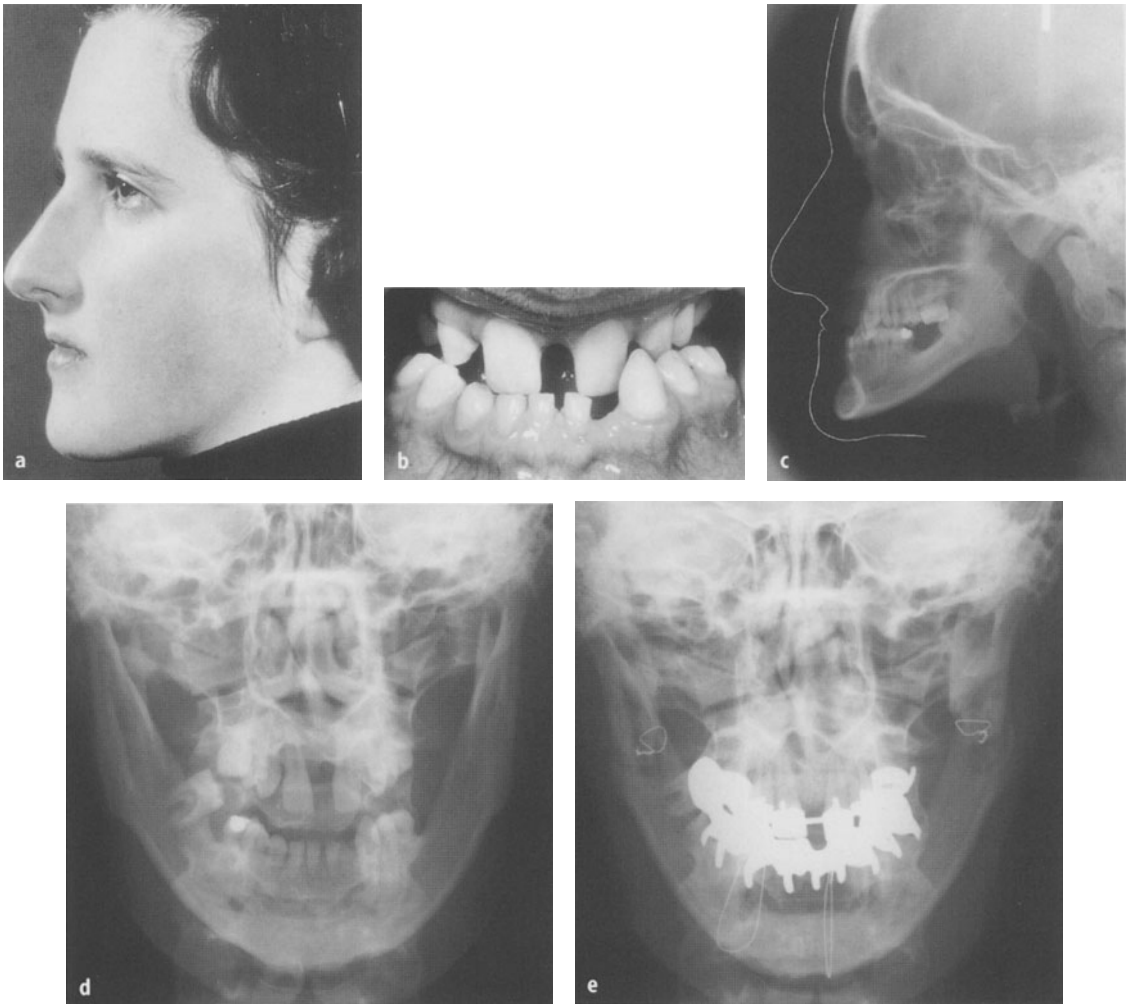


Fig. 83a–e. First case of sagittal splitting of the rami procedure, performed under general anaesthesia (9 April 1956). **a, b** Patient's pre-surgical profile view and occlusion. **c** Presurgical lateral cephalogram. **d, e** Mandible p.a. projection before surgery disclosing rather slim rami and 10 days after surgery, with cemented and additionally fixed cast cap splints

I then fixed the mandible in the preplanned occlusion. On the first side I fixed the fragments in the new position with the then usual anterior border wiring. I closed that side. I had great problems with the second side. The proximal and distal fragments had no contact. Finally I managed to adapt the free lateral ramus fragment to the rest of the ramus and the main fragment, using direct wire fixation (Fig. 83e). Then I closed the wound and inserted a small rubber drain as I had always done before. The operation took me over 4 h. I thanked God that it was over and hoped for the best. The patient received penicillin.

The operation itself was more stressful than I had experienced previously when I had performed it with the patient under local anaesthesia and general sedation. In

addition, I had to battle with the anaesthesiologist and my assisting pessimistic chief during the surgery. The former was concerned that I would pull the tube out of the patient's nose by turning her head.

On the second postoperative day and even more on the third, the girl's face was swollen and blue from bruising so much that I feared serious complications. On the following day, I went to the wonderful Baroque monastery church of Einsiedeln and prayed, and promised I would never do a sagittal splitting again if that girl got away without complications. Finally she had a wonderful result and I became a recidivist, like any sinner, doing many more cases. To complement her improved appearance, her dentist managed to solve her occlusal problem rather nicely, aesthetically as well as function-

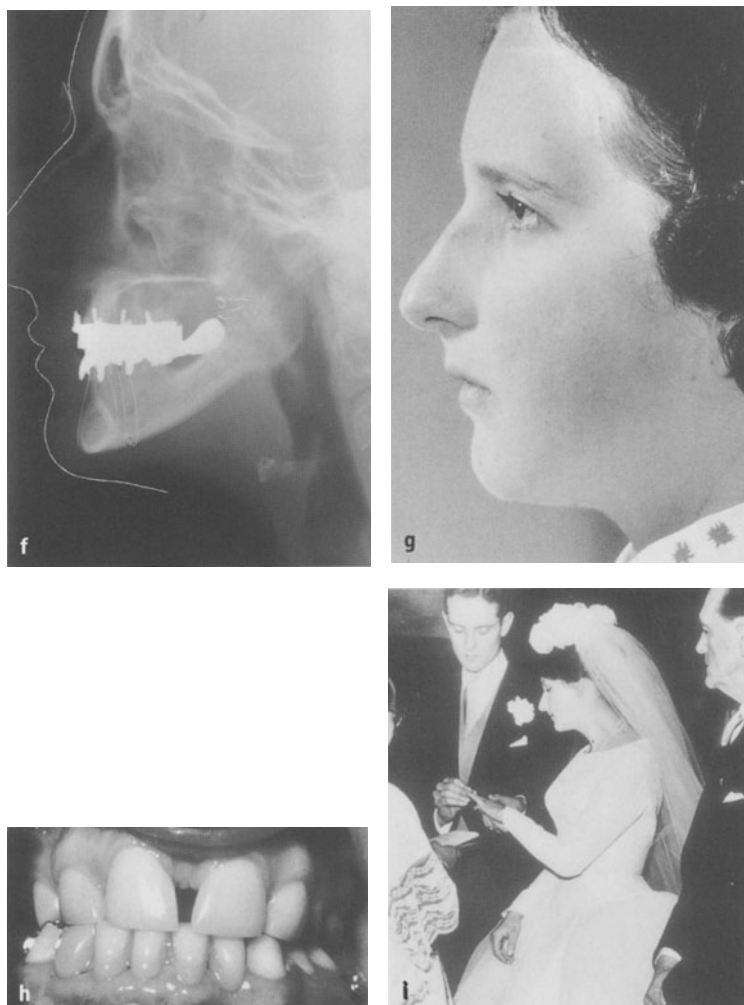


Fig. 83f–i. First case of sagittal splitting of the rami procedure, performed under general anaesthesia (9 April 1956). **f** Lateral cephalogram 10 days after surgery. **g, h** Patient's profile and occlusal situation 5 months after her mandibular set back operation. **i** Patient's appearance 6 years after surgery

ally (Fig. 83f–h). Six years later the girl sent me her wedding photograph. It proved that the surgery was successful and there was no sign of relapse (Fig. 83i).

No doubt an advancement of the maxilla in addition to the set back of the mandible would have done some additional good to the patient's profile. Maxillary advancement did not then exist. It was still some 6 years before I had developed it as a standard procedure.

When for the first time I read a paper on this sagittal splitting procedure on the rami at the annual meeting of the German Association of Maxillofacial Surgery and after I had finished, the professor presiding at that lecture jumped up from his chair and said: "What this young man is suggesting to us is not surgery, it is filthy. We have learned by experience that we must abandon a

bone grafting operation to the mandible, when by chance the oral mucosa is perforated, even by the width of the head of a pin only".

In retrospect, it was worthwhile to go through all the trouble in developing this procedure and improving it to an easily learnable standard technique. With this and other procedures I learned the truth of my principle which says: "*It is not difficult to find a solution to a problem, it is only difficult to identify the problem*" (Principle No. 35). My teacher, R. Trauner, had recognized this problem. So it is to his credit that I got the idea of developing the sagittal splitting of the rami procedure.

The problem with naso-tracheal intubation and therefore with the anaesthesiologist I have experienced rather often, even more seriously when I did demon-

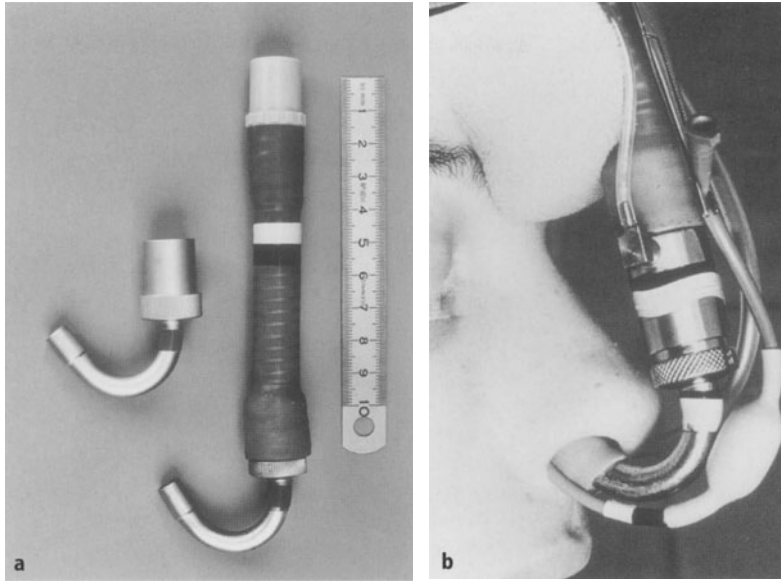


Fig. 84a,b. Special nasal intubation arrangement for prolonged surgery in the mouth. **a** Specially curved connecting piece with elongation tube to prevent damage to the nostril during prolonged craniofacial surgery and surgery in the mouth. **b** Nasal intubation arrangement on the operating table

stration operations in other countries. Later, I carried all the necessary special equipment with me, even a specially curved connecting piece for the tube (Fig. 84a,b), made by Medicon, Tuttlingen, Germany. For long opera-

tions with naso-tracheal intubation, that connecting piece is a blessing for the patient's nose, and for the surgeon! Today I still prefer it to the currently-used curved plastic intubation tubes.

20.4

Modifications and Further Development of the Procedure

With increasing experience, I and others modified the procedure for additional improvements. The intention was to produce longer, raw (cancellous) bone surfaces

for better and earlier bony union and to accommodate a wider range of movement of the jaw after the ramus has been split (Fig. 85a).

20.4.1

Dal Pont's Alteration of the Direction of the Lateral Cortex Cut

After the procedure was published in the English language literature (R. Trauner and H. Obwegeser 1957), including photographs taken of the various steps taken during the operation, international interest in this procedure started to develop. When Dr. Dal Pont came to Zürich as a trainee to Prof. Schmuziger, who was our chief, he assisted him and me with all cases. From watching, he conceived the idea of directing the lateral cortical cut from the second molar region vertically down to the lower border instead of towards the angle as I did. Prof. Schmuziger and I tried it that way with Dr. Dal Pont assisting us. It worked. He made a drawing and called it "sagittal retromolar osteotomy". He also

noticed that in some cases the split did not go all the way back to the posterior border. He made a drawing of this and called it another new procedure of his "oblique retromolar osteotomy". He then published both drawings as two new procedures (G. Dal Pont 1959, 1961) without mentioning that the sagittal splitting of the ascending rami already existed as a procedure (R. Trauner and H. Obwegeser 1955, 1957). He also did not mention that the case which he published (without permission) was not operated upon by himself but by me. Even up until now, he has never personally performed a sagittal splitting procedure, as he admits. However, his ideas were useful.

20.4.2 The Incomplete Sagittal Splitting of the Ascending Ramus

With Dal Pont's alteration of the direction of the lateral cortical cut down to the lower border we do get longer contacting bone surfaces in the horizontal ramus. This is required when advancing the mandible rather far forwards. But it is not necessary, usually not even in the case where the sagittal split passes incompletely behind the lingula down in front of the angle, called by Dr. Dal Pont "oblique retromolar osteotomy" and also suggested by E. Hunsuck (1968) and B. Epker (1977) (Fig. 85b), because the amount of cancellous bone still comprises almost two-thirds of the breadth of the ascending ramus. I myself call this the "*incomplete sagittal splitting*" of the ramus. This is fine for advancing the mandible when the lateral cortical cut goes down to the lower border making up for the diminution in contacting cancellous bone surface. But the incomplete sagittal splitting of the ra-

mus together with the lateral cortical cut down to the lower border has several disadvantages when compared with the complete sagittal splitting of the ramus with the lateral cut towards the angle (H.P. Freihofer 1976):

1. More risk to the mandibular nerve as it comes closer to the lateral cortical plate in the molar region than in the angle area. We did over 500 splittings that way and then directed our lateral cortical cut towards the angle again (M. Farmand and H. Obwegeser 1981) because of too high an incidence of damage to the mandibular nerve (W. Peppersack and J.M. Chausse 1978) and for the following reasons.
2. It eliminates the possibility of improving a flat angle.
3. A very important disadvantage with the incomplete sagittal splitting of the ramus is the fact that for the push back operation, the cancellous bone of the main fragment will overlap the cortical plate of the proximal segment as it does not permit the major segment to slide back without pushing the ascending ramus outwards (Fig. 85c).

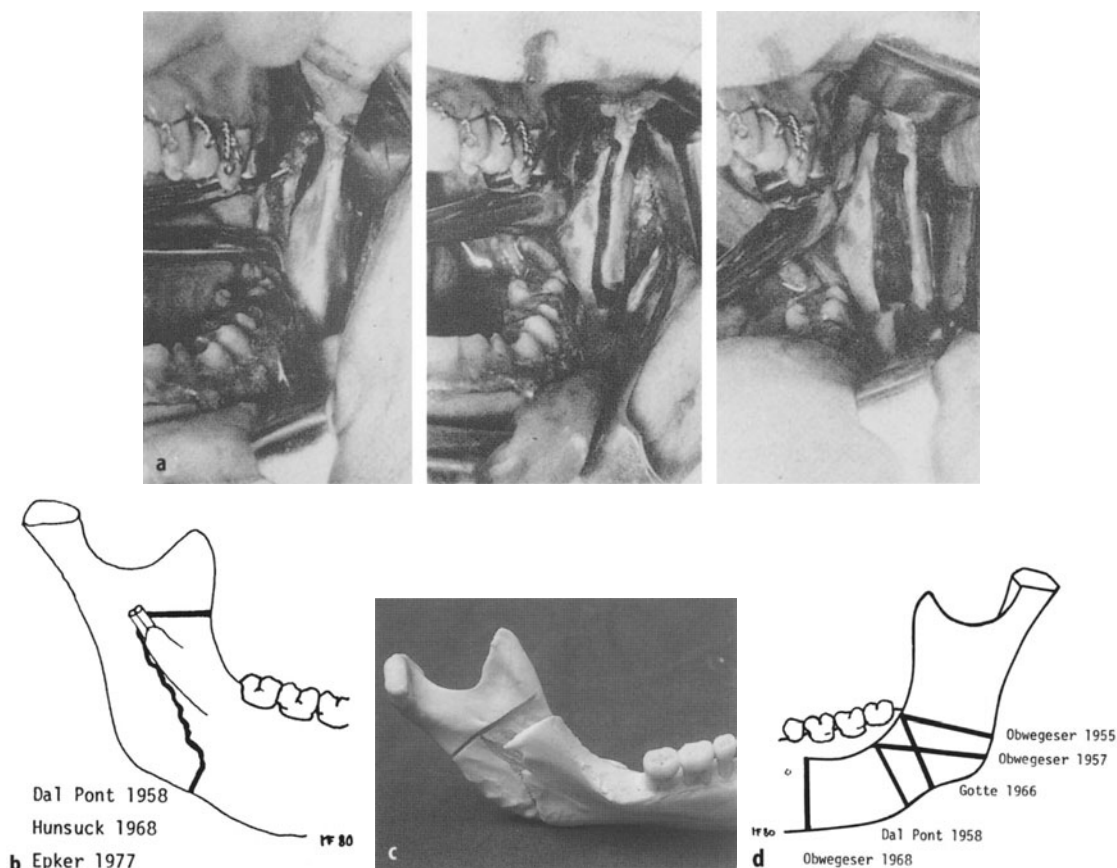


Fig. 85a–d. Modifications and further developments of the sagittal splitting procedure. **a** Steps of the operation of elongated sagittal splitting of the ramus (from H. Obwegeser 1957). **b** Incomplete sagittal splitting of the ramus procedure (from M. Farmand and H. Obwegeser 1981). **c** After incomplete sagittal splitting, the proximal fragment is pushed laterally when the mandible is repositioned posteriorly. **d** Modifications of the location and direction of the lateral cortex cut (from M. Farmand and H. Obwegeser 1981)

4. With the lateral cortical cut directed to the angle or even slightly above it, the splitting all the way to the back of the ascending ramus is much easier. The bone segments can slide along each other much more satisfactorily than with the elongated part of the horizontal ramus on the proximal fragment, which rather often also has a certain degree of curvature. Because of that curvature, an empty space between the two segments will exist when the jaw is repositioned.

With the exception of the incomplete splitting of the ramus which may happen accidentally, but should not be intentional, there were no other alterations suggested on the lingual aspect.

For splitting the ramus, some people prefer to perform it using a saw (T. Kitajima et al. 1989). If they do not damage the nerve with that instrument it is acceptable.

20.4.3

The Long Lateral Surface Splitting of the Horizontal Ramus

On the lateral aspect of the ramus and the preangle area, several locations and directions of the lateral cortical cut have been suggested (Fig. 85d). For the set back operation of the edentulous antemandibulism case I wanted to dispense with the acrylic splints fixed to the jaws because of their inconvenience to the patient caused during the period of intermaxillary fixation. For that reason I used a long lateral surface split as far forwards as the mental foramen. That permitted fixation of the fragments together, without intermaxillary fixation (H. Obwegeser 1968a). At that time no screws or plates were used. One direct wire at the anterior border of the ramus and a circumferential wire in the body region was enough (Fig. 85e). One has to be very careful not to damage the mandibular nerve as it can run really very laterally in the horizontal ramus. I modify the localization of my lateral bone cut according to the needs of the case. For severe retromandibulism cases and in particular for the bird-face appearance I always use a long-surface split (see Fig. 13n, o).

20.4.4

The Sagittal Splitting Procedure for Elongation of the Mandible in Its Horizontal and Vertical Dimensions

The sagittal splitting procedure of the ramus not only permits alteration of the length of the horizontal ramus and the angle. It also includes the possibility of elongating the length of the ascending ramus. This happens automatically in those cases in which the maxilla has to be

lowered. This is true in all cases in which a unilateral maxillary hypoplasia results due to hypoplasia of the condyle when acquired in early childhood or even in infancy, independent of the cause. It is particularly a fact in the case of unilateral ankylosis occurring at a very early age (see cases Figs. 22 and 23). Elongation of the ascending rami may also be desirable for aesthetic and prosthodontic reasons in bilateral cases without moving the maxilla inferiorly.

One of the cases of most severe bird-face appearance I had to deal with was due to a bilateral ankylosis since birth. The child had been a forceps delivery. Immediately after birth, a tracheostomy was performed because of the inability of the child to breathe. The patient could never open her mouth and could only take liquid food until age 18 years when US soldiers brought her from their military base in Italy for evaluation and treatment. They also generously supported her hospitalization expenses.

In a first intervention, I released the bilateral ankylosis, using our standard procedure (H. Obwegeser and M. Farmand 1982). It took quite some time to stretch the musculature so much that a 40 mm interincisal distance was achieved during surgery. The gap in the TMJ region was filled with a pack of thin slices of lyo-cartilage, as always. Supervised regular mouth opening exercises were carried out for a full year with an acrylic interdental splint worn during the night.

In a second intervention (Fig. 85h) the protruding maxillary front segment was repositioned posteriorly and a long surface splitting of the mandible permitted us to increase the length of both the horizontal as well as the ascending rami and a double-step chin advancement plus an additional piece of cortical bone as a third step elongated the mandible in the anterior region. The result was very pleasing (Fig. 85i, j). When I saw the patient again by chance some 12 years later, there was some degree of loss of horizontal length noticeable. However, the case proved that the sagittal splitting procedure permits us to elongate the mandible in its horizontal as well as vertical dimension. It proved another interesting fact: the quantity of skin for an elongation of 40 mm is available. It only has to be mobilized.

As an alternative to the possibility of directing the lateral bone cut vertically to the lower border somewhere between the angle and the mental foramen, I have also seen a former trainee of mine (A. Triaca) producing a cut closely parallel to the lower border. With that he seeks to prevent the splitting all the way down in severe retromandibulism cases. Otherwise the gap in the lateral cortical plate produced by the advancement of the mandible may become visible. With his horizontal osteotomy above the lower border he attempts to avoid that problem.

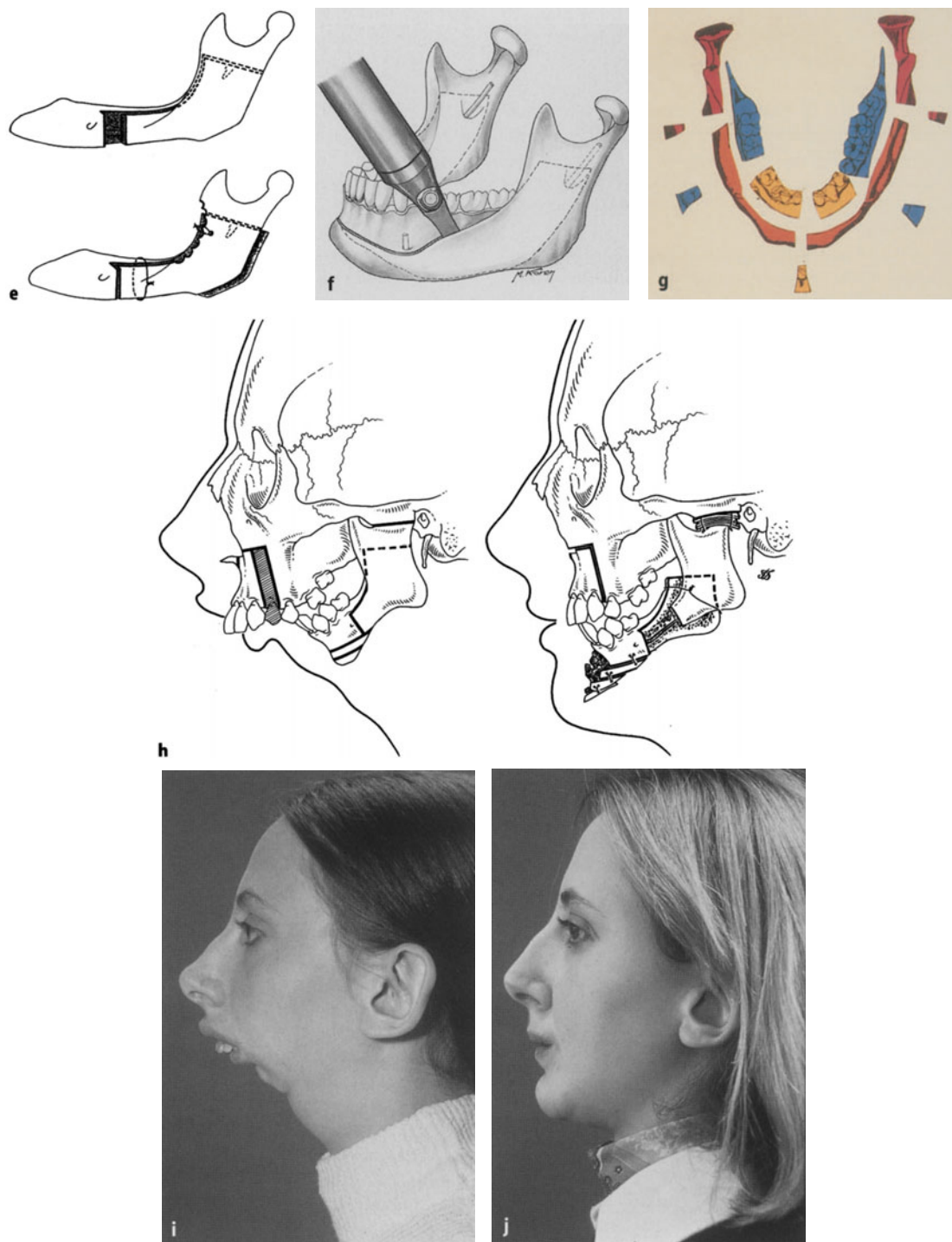


Fig. 85e–j. Modifications and further developments of the sagittal splitting procedure. **e** Long surface splitting for correction of an edentulous antemandibulism (from H. Obwegeser 1968). **f, g** The circular splitting of the mandible according to J. A. Obwegeser (1987, 1988). **h** The sagittal splitting procedure for elongation of the mandible in its horizontal and vertical dimensions applied in an extreme bird face appearance case together with triple-step chin advancement and correction of maxillary protrusion (from H. Obwegeser 1971). **i, j** The patient before and after one intervention for correction of the severe bird face appearance (from H. Obwegeser 1971)

20.4.5

The Circular Splitting of the Mandible

(J. A. Obwegeser 1987, 1988)

My nephew, J. A. Obwegeser (1987, 1988), has extended the sagittal splitting all the way around the mandible. In the mental foramen areas bilaterally he leads the lateral cut inferiorly around them (Fig. 85f). I call this the *circular splitting of the mandible*. This technique produces two horseshoes of the mandible, the inner one carrying the teeth. It provides the possibility of raising the arch of the teeth for the correction of a deep overbite. In addition, the outer and the inner horseshoe of the split mandible can be segmented for the desired corrections. From the tooth-bearing horseshoe-like lingual part, a tooth including its bone can be excised, or a gap in the row of teeth can be closed by excision of the bone where a tooth is missing (Fig. 85g).

20.4.6

The Transoral Angle Osteotomy

(H. Obwegeser 1964)

By experience I had learned that there are limitations in moving the mandible posteriorly and also additionally in rotating the mandible adequately in severe open bite cases. For these cases I wanted a procedure which leaves the ascending ramus with its musculature and ligaments in position, but permits closure of a severe open bite with an osteotomy in the angle area and simultaneous repositioning of the mandible posteriorly or anteriorly by a large amount. The sagittal splitting procedure gave me the idea of performing an osteotomy at the angle region after the lateral cortical plate has been detached from high up and far forwards. Once the mandibular nerve is exposed and freed, the osteotomy in the angle region is easily performed. When the jaw is fixed

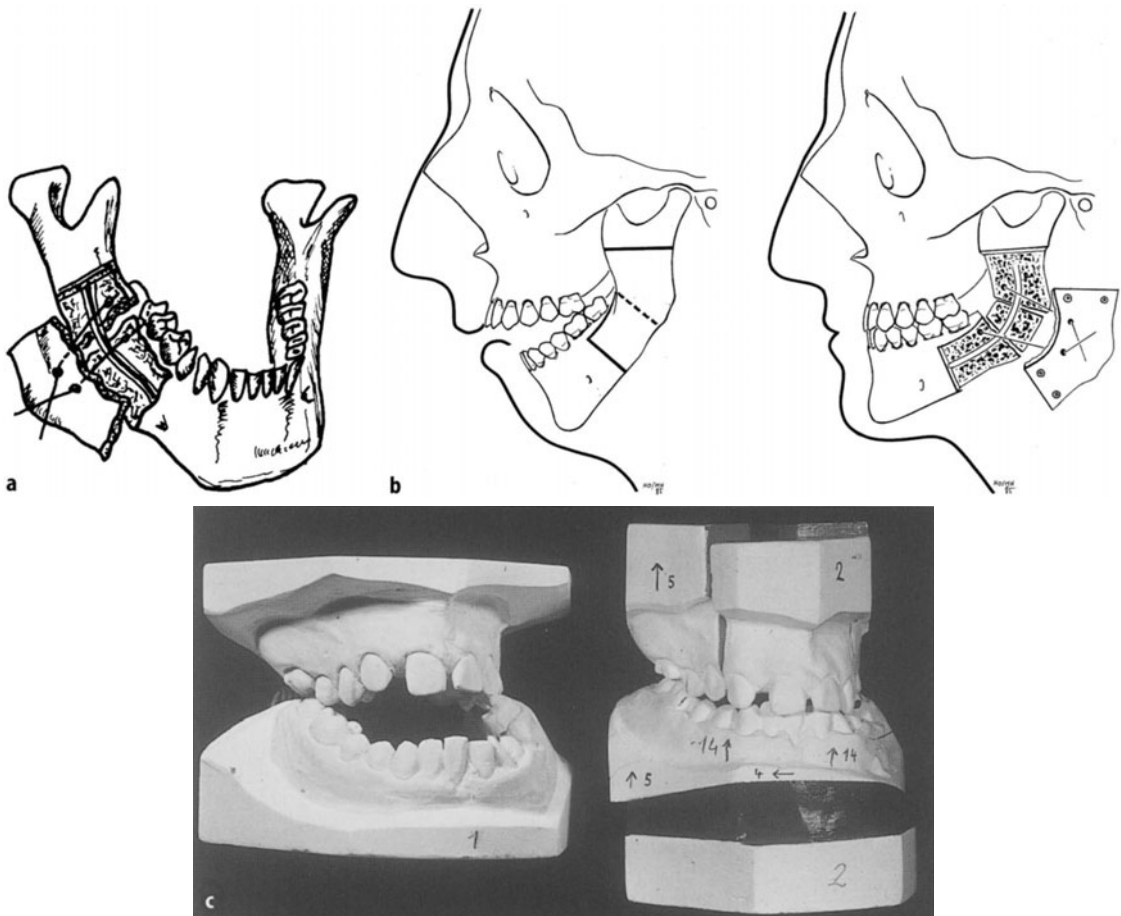


Fig. 86a–c. The transoral angle osteotomy procedure (H. Obwegeser 1964). **a** Schematic illustration of the procedure derived from the sagittal splitting technique (from H. Obwegeser 1964). **b** Illustration of the technique for elongating the mandible in the angle region. **c** Model operation for correction of severe open bite case

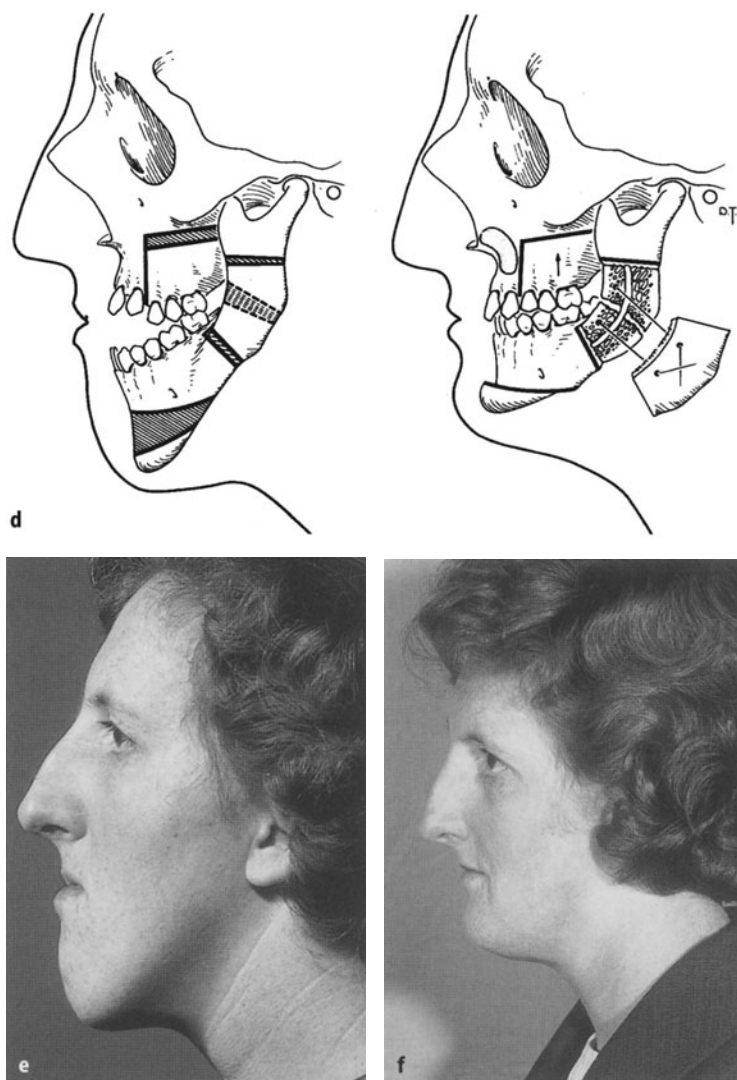


Fig. 86d–f. The transoral angle osteotomy procedure (H. Obwegeser 1964). **d** Schematic illustration for the correction of this very severe open bite case. **e,f** The patient's presurgical profile and 1 year after surgery (from H. Obwegeser 1964)

in the planned occlusion, an overlapping part of the lingual section at the angle area is excised and the lateral cortical plate is repositioned and fixed. It will bridge any remaining defect on the lingual aspect of the angle. If there is a larger gap because the mandible had to be advanced by quite an amount, the lingual defect may be filled with a piece of bone. This procedure can be used alone (Fig. 86a,b) or in combination with others.

This case of bimaxillary long face anomaly of unknown origin presented such a complex problem. The mandible was much too long compared with the maxilla. It had a very stretched angle and a chin much too high (hypergenia). Apart from this mandibular anomaly, the posterior parts of the maxilla were too far down, thus increasing the degree of open bite.

The model operation planning showed the necessity for bimaxillary intervention (Fig. 86c). It was only possible to achieve an acceptable occlusion and aesthetic result with an additional repositioning of the posterior maxillary segments higher up, in addition to the necessary correction of the anomalies of the mandible. For the latter I decided to use the angle osteotomy procedure in this case, as it permits unlimited rotation and simultaneous shortening in the angle region. The combined intervention on the maxilla as well as on the mandible is demonstrated by the schematic illustration shown in Fig. 86d. With that type of combined surgical correction of the maxillary and mandibular deformity, the planned result was achieved (Fig. 86e,f).

20.5 My Technique For Many Years

The occlusion is prepared presurgically, in such a way that the two jaws can be brought together in an acceptable occlusion, if necessary with the use of a splint. Nasotracheal intubation anaesthesia is so arranged that it does not interfere with the surgery in the mouth (see Fig. 84).

Before surgery starts a cortisone derivative is injected or any other medication is given which will reduce postoperative swelling. This is repeated after surgery or 3–4 h after the first medication. For reduction of bleeding during the surgery and of ecchymosis, either a vasoconstrictor is injected into the operation fields or the blood pressure is lowered.

In order to avoid complications during surgery, or maybe even later, proper modern instruments are necessary. The less traumatically we operate, the less postoperative swelling and problems will we have. In order not to damage the lips, I prefer the gutter-shaped retractors. For retracting the cheek mucosa I want those with the upwardly curved end (see Fig. 102b) while for the subperiosteal retraction I do not mind those which are bent downwards at their end.

I always start on the abnormal side, if there is one more so than the other. A rubber mouth gag on the other side holds the mouth open.

The Incision. The incision in the mucosa starts laterally, from the first molar, well into the moveable mucosa, vertically through it and then through the periosteum between the buccinator crest and the oblique line thus avoiding the facial artery. It is extended upwards along the external oblique line to the base of the coronoid process. Then the periosteum is elevated in the retromolar region and from the anterior border of the ascending ramus and a few millimetres on either side of it all the way up, including inserting fibres of the temporalis muscle. For that I use the swallow tail periosteal elevator. Once the anterior border is freed I clip it with the ramus clamp as high up as possible. It prevents the soft tissues from coming down again and its shield holds the cheek out of the operating field.

The Cortical Cut above the Lingula. Next, I elevate the periosteum on the lingual side between the lingula and the semilunar notch only as far back as to the concavity above the entrance of the mandibular nerve into the foramen. Before I start with the bur I check the semilunar notch using a Freer-type periosteal elevator. I want to be sure that I am not below the lingula but below the semilunar notch. Then I use an acrylic bur to start my lingual cortical cut. It makes a broad roundish defect in the wide anterior border. This permits better vision into that difficult lingual area (Fig. 87a). Then I raise the rest of the periosteum all the way back to the posterior bor-

der and around it. Next, to keep the lingual soft tissues away and to protect the mandibular nerve I like the narrow mandibular channel retractor, inserted and hooked behind the posterior border. Under direct vision one can now elongate the lingual cortical cut all the way back including the posterior border.

As far as the cortical cut above the lingula is concerned I have given up using the Lindemann bur, not only because it breaks easily but also I want to be able to shorten the ascending ramus length on its medial side, particularly in a push back operation. For that reason the Lindemann bur makes too narrow a cut. With today's long, special surgical handpieces (see Fig. 102a) for the electric motor, we can use any convenient bur with a shaft of 2.35 mm diameter to make a broad cut further back. My cut with a special bone cutting bur¹, preferably 2.9 mm in width or any other similar bur even up to 5 mm diameter, goes only so deep that I can see bleeding points from the cancellous bone all the way back. For that, the lingual bone cut must not be too high up near the semilunar notch.

Above the lingula I want a rather broad defect in the medial cortical plate of the ramus in order to prevent overlapping of the main fragment over the inner cortical plate of the proximal fragment. I generally prefer the splitting of the whole ramus breadth. Otherwise the cancellous part of the main fragment will overlap the cortical plate of the proximal fragment, pushing it laterally (see Fig. 85c) when the mandible is repositioned posteriorly. I also like it for the correction of a retro-mandibulism case, although in such a case it does not matter that much.

The Buccal and the Connecting Cortical Cuts. Next, I elevate the periosteum on the buccal side of the ramus where I want to place my lateral cortical cut. I free it around the lower border. Care should be taken that the masseter muscle is left attached to the lateral aspect of the ramus, as suggested by W. Bell and S. Schendel (1977), not only for better blood supply but also for better stabilization of that proximal segment. In an ordinary set back or advancement operation I normally direct that cut somewhere to the angle area, starting in the second molar region (Fig. 87b). I can feel the angle of the jaw with my periosteal elevator. The periosteum including the masseter muscle is raised along the line of the lateral cortex cut and around the angle. I prefer to direct my lateral cortical cut towards the angle for three reasons:

1. Firstly, in that area the mandibular canal is not so far lateral as further forward
2. Secondly, when a correction of the angle is desired then the lateral cortical cut in the angle area is necessary

¹ Maillefer S.A., CH-1338 Ballaigues, Switzerland

3. Thirdly, in the angle area there is much less risk of damaging the facial artery and the mandibular branch of the facial nerve than is the case when the bone cut passes vertically down to the lower border.

That lateral cut varies in my surgery according to each individual case. In a case of micromandibulism such as a bird-face appearance it will reach almost as far forwards as the mental foramen, starting behind the second bicuspid (see Fig. 13n, o).

In every case, the broader mandibular channel retractor is hooked around the lower border to protect the soft tissues. Without it there, it is not difficult to cut the facial artery or even the mandibular branch of the facial nerve with a bur or saw when the soft tissues are not properly protected. Again I use a 3 mm diameter rotating bone-cutting bur, going only as deep as necessary to see bleeding points from the cancellous bone all the way down. The cut through the cortical plate has to pass almost around the lower border. When that bone cut is completed, next the lingual and the lateral bone cuts are connected. The vertical connecting cut follows the anterior border just on its inner side, using a rather narrow bone-cutting bur or an oscillating saw. In order to avoid the bur slipping around the anterior ramus border it is helpful to drill holes with a small rosehead bur along the medial side of the ramus edge. Care must be taken that the cutting instrument goes through the cortical bone only, no deeper than 4–5 mm.

During all cutting work the cutting instrument must be irrigated with copious amounts of saline solution in order to avoid clogging and, as a consequence, burning the bone. Before the actual splitting I rinse the operating field with an antibiotic solution.

Splitting the Ramus. For the splitting itself, G. Smith had a good idea with his spreader instrument. I personally like to open the vertical cut by 2–3 mm with a wedge osteotome first (Fig. 87c). Then I insert my bone separator, but only a few millimetres deep (Fig. 87d). Its fork-like blades are flexible. With that instrument the splitting is done very gradually and gently, waiting a few

seconds after each incremental step. If the ramus does not want to split easily a blow with a thin osteotome on the inferior border will help. Thereby, the separation of the two cortical plates occurs gradually until the two halves all of a sudden fall apart all the way back (Fig. 87e).

When the lingual cortical cut is not deep enough all the way back to the posterior border then an incomplete splitting will occur, just behind the lingula all the way down, leaving at least one-third of the ramus breadth unsplit (see Fig. 85b). If the cortical plates do not want to split at the inferior and posterior border, their wide separation by the bone separator permits visualization of the mandibular nerve between them. As the bone separator can be fixed in its open position, the nerve can easily be freed under direct vision when it, or even the whole canal, adheres to the proximal fragment. Once the nerve is completely free, it is possible to cut the posterior border with a thin osteotome or a bur, without any risk to the mandibular nerve as it can be visualized and protected with the bone separator in place. The use of the bone separator also permits trimming off the cortical unevenness of the ramus fragments or even the posterior border, under direct vision.

When both sides are split the mandible can be moved forwards, backwards, sideways or upwards, depending on the need (Fig. 87f). The mandible is then fixed in the planned occlusion, either by holding it there, or better, by using some intermaxillary wires.

Nibbling off the protruding rim of the proximal segment of the ascending ramus is often necessary when the main segment has to be repositioned posteriorly. The amount of shortening of that anterior rim depends on the amount of posterior repositioning of the main segment and the preoperative direction of the ascending ramus. In a case of severe H.E., slender type, which also requires improvement of the angle region, a part of

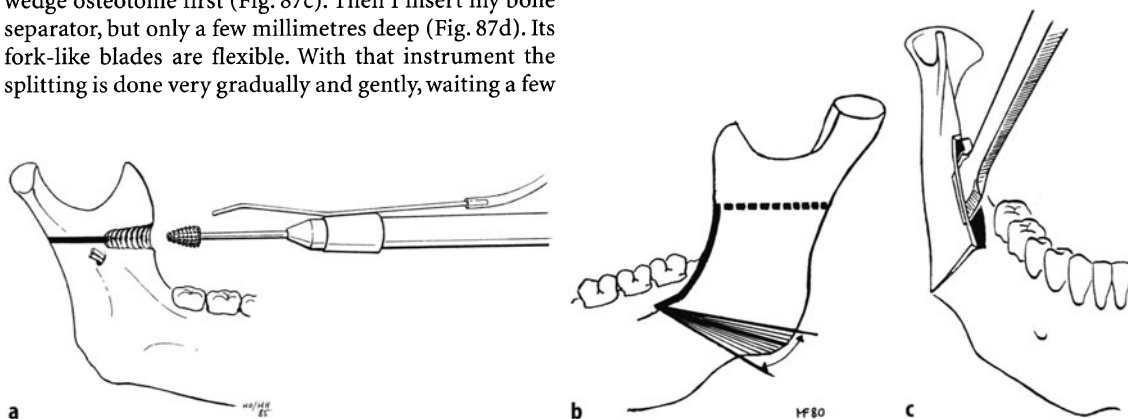


Fig. 87a–c. My technique for sagittal splitting for many years. **a** The first step on the lingual side of the ramus. **b** I place the lateral cortical cut somewhere in the angle region. **c** The splitting is commenced with the wedge osteotome

that amount protruding can be reduced by rotating the proximal fragment further back. This is also required to produce a better angle. However, the posterior rotation of the proximal fragment has some limitations because of the joint and because thereby the upper part of the protruding rim will protrude even more. So, in a severe H.E., slender type, case requiring a large amount of pos-

terior repositioning of the mandible for occlusal reasons, the amount of necessary reduction of the anterior rim may be quite substantial.

The little pieces of bone obtained during that procedure may be needed to fill a gap between the inner and outer cortical plates in order to prevent their being forced together when placing the screws.

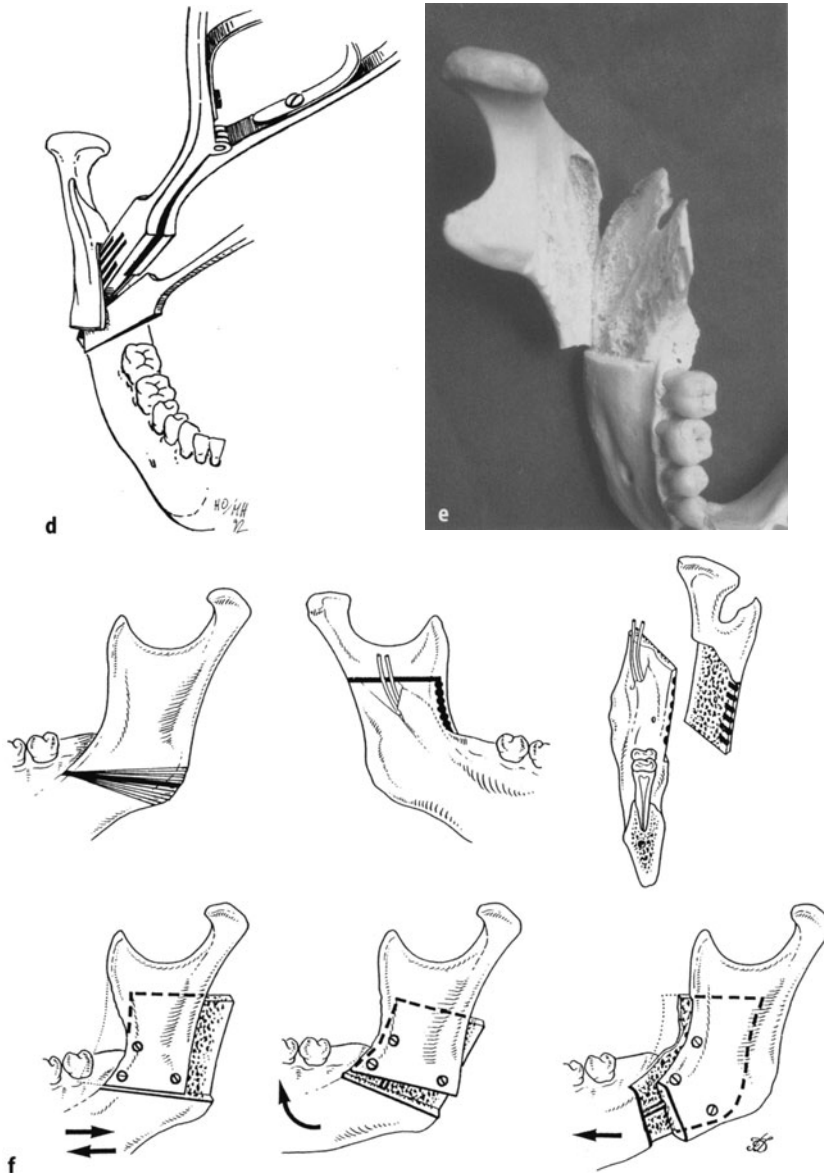


Fig. 87d-f. My technique for sagittal splitting the ramus for many years. **d** The bone separator is inserted in the splitting line and gently opened. When the inferior border does not seem to want to split, a blow on a sharp osteotome will help. **e** Splitting of the ascending ramus all the way back to the posterior border. **f** After the splitting, the mandible is freely movable in any desired direction, still ensuring large contacting bone surfaces

The Osteosynthesis. The osteosynthesis is, nowadays, always done by lag screws or by the use of plates and short screws. I like lag screws as there is plenty of room to place them safely when splitting of the full width of the ramus has been performed. With the mandible fixed in the desired occlusion, the condyle on the proximal ramus fragment is manipulated into the glenoid fossa and held with an adaptation clamp, or any other suitable instrument in that position by the assisting nurse or doctor.

It is my experience that almost 50% of the patients show a slight occlusal imbalance after recovery from general anaesthesia. The conclusion is that under the effects of the muscle relaxant, the surgeon places the mandible into a different occlusion to that which the patient's musculature places it spontaneously.

In order to secure the preoperative position of the condyle H. Luhr in 1985 and 1986 recommended that before the sagittal splitting of the ascending ramus, the ascending ramus be connected to the maxillary splint or the maxilla itself, with a bar and screws (Fig. 87h). During the actual splitting of the ramus, the device is removed again but is reapplied before screws or plates fix

the fragments in their new position. In cases of bimaxillary osteotomy a bar should connect the ascending ramus with the zygomatic crest of the maxilla, above its osteotomy line (H. Luhr 1989, 1995). Thereby the preoperative position of the condyle is guaranteed and the possibility of occlusal inaccuracy after the patient awakes from the general anaesthetic is usually eliminated.

Then I drill two 1.2 mm diameter pins transbuccally through both cortical plates. Using these, the ramus fragments are stabilized enough for the insertion of the screws without the risk of the fragments changing position while inserting screws or plates for osteosynthesis (Fig. 87g). Then the pins are pulled out again, one after the other, using the holes in the cortical plates for inserting a bur or the direct use of self-tapping screws of 1.5 mm diameter. Each surgeon has his own preferences: I always use three screws on each side and without compression. I often wondered why surgeons use screws of so thick a diameter as if they were operating on a horse. A diameter of 1.5 mm with a head of 2.0 mm will be strong enough, even when titanium screws are used.

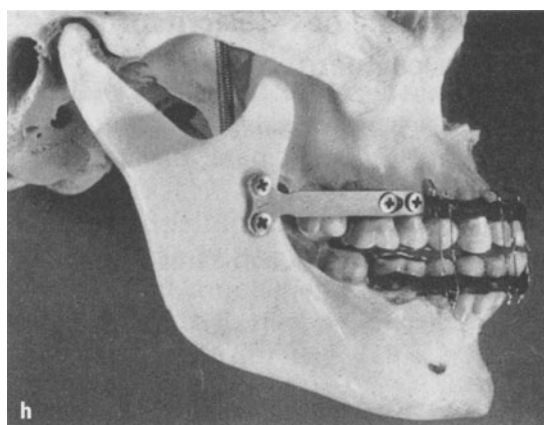
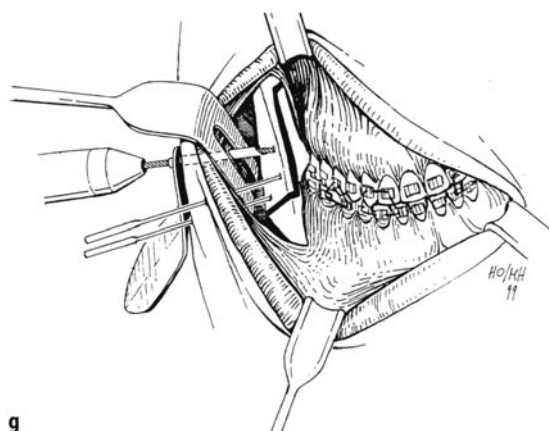


Fig. 87g–i. My technique for sagittal splitting for many years. **g** Stabilization of the two fragments for screw fixation, by two transbuccal pins. **h** Ensuring the presurgical position of the condyle by a bar which connects the proximal segment to the maxilla or zygoma before and after the splitting (with permission of and gratitude to H. Luhr 1985). **i** Both proximal segments have rotated upwards after accidental horizontal osteotomy above the lingula, producing an open bite after bony union with the distal segments (from a referred case)

There are many different types of screw head on the market. I prefer the central locking type in which the screwdriver fits so tightly that with it the screw can be handled. That means, that a cannula with a lumen of 2.1 mm diameter is large enough to let the bur pass and then the screwdriver with the self-tapping screw on it, through the cheek to the mandible. A split-(fork-)retractor holds the soft tissues away for direct vision of the operating field and simultaneously permits the bur or the screwdriver in the cannula to reach the bone (see Fig. 87g).

It is absolutely contraindicated to compress the proximal fragment to the mandible by force. By doing so the mandibular nerve can be damaged. In order to prevent this, some surgeons drill a channel for the nerve where it is finally expected to be. Also, by forcing the fragments together the condyle may be pushed out of the glenoid fossa. The main reason for this may be that the distal fragment is overlapping the cortical covering of the proximal fragment, either as a consequence of incomplete splitting, or even above the lingual cut. These impediments should be trimmed off. If a gap still remains between the two ramus plates some small pieces of bone should be placed in between them to ensure that the lateral fragment is not pulled towards the lingual one. These small pieces of bone are automatically acquired during the necessary nibbling off of the protruding anterior rim, either of the lateral or the medial ramus fragment, depending on whether the mandible has to be positioned forwards or backwards.

Any screw could do some harm to the nerve or a tooth root, although this is very unlikely if the necessary care is taken with screws of small diameter. In order to get around even this possibility, plates may be used for stabilizing the fragments in the desired position. As I do not have any experience with this method I am unable to say whether or not it can be recommended.

Before closing the wound with a continuous non-resorbable monofilament suture, the operation field is again rinsed with an antibiotic solution. I like to place a

thin rubber drain in the operation field for 1 day, but no vacuum drainage, particularly not after operating under hypotensive anaesthesia.

For removal of the screws later, no transbuccal cannula is necessary. Via a very small stab incision the screwdriver can be passed through the cheek to the screws after the lateral aspect of the mandible has been freed transorally. In this way the screws can be removed via the oral route.

Advantage of Resorbable Screws and Plates. As mentioned earlier, there is always progress in techniques and material. Some authors make a case for not leaving any foreign material in the body. They want even titanium screws and plates removed. Consequently, screws and plates made from synthetic resorbable materials are advocated. J.A. Obwegeser (1994a,b) advocates screws and plates made from allogenic cortical bone. He could prove experimentally as well as clinically that they are strong enough and that they become substituted by local bone tissue in a shorter period of time than do the synthetically produced screws, and contrary to them, without any signs of local inflammatory reaction.

I consider the use of resorbable screws and plates for osteosynthesis a great advantage compared with metal screws if we think of the possible necessity to remove metal screws again for whatever reason, particularly when after some time or even years a sagittal splitting becomes necessary again. Regrettably they still need to be of a larger calibre than the metal screws.

Looking back at the beginning of the sagittal splitting procedure, I almost envy those who were able to learn it after it had been perfected and can use the special instruments that are now available. According to my experience, the sagittal splitting technique has become a very safe procedure without any risk at all if the surgeon knows his job and uses the suggested special instruments. It would have been easy to develop it, even under local anaesthesia, with the help of today's instruments.

20.6

Principal Complications and How to Deal with Them and How to Avoid Them

Naturally, I have been confronted with many problems in connection with the sagittal splitting procedure. Some of them I have experienced myself, some others through the many trainees I had at my clinic and quite a number also through phone calls or referral of patients with complications.

The complications depend basically on the experience of the surgeon and the type of instruments used or because the proper ones were not available. The most common and the most dangerous complications I will discuss here.

Problems During Surgery

These can arise quite easily, for several reasons.

On Incising the Mucosa. The incision along the ascending ramus can unexpectedly run into *massive bleeding due to a perimandibular haemangioma*. Ligation is impracticable, packing only (see case of Fig. 20) is required.

The Facial Artery Can be Damaged. It will cause excessive bleeding. Extraoral ligation is the method of choice. I found it very difficult to clip it through the incision opening.

Damage to the Facial Nerve. There are several possibilities. The mandibular branch of the facial nerve can be damaged by the assistant's retractor. Spontaneous recovery after some weeks or months may be expected, if possible supported by electrotherapy.

The mandibular branch may have been cut by the saw or the rotating bone cutting instrument. It will probably not be diagnosed before the patient is awake again. When the nerve has been cut resuturing is mandatory.

The whole facial nerve may lack function after surgery. This has also been reported. It is unlikely that the main nerve trunk was cut, just damaged by traction on the assistant's retractor. Spontaneous recovery should be anticipated. It might be wise to consult a neurologist in such a case. This is also true if complete (idiopathic) facial palsy occurs without any direct mechanical damage to the nerve itself. I have also heard of this complication.

The Lingual Cortical Cut Can Produce Several Problems. A fracture of the ramus at that level may occur when cutting the lingual side. The burr or the saw can pass almost all the way through to the buccal surface. When extending the lingual cut to the posterior border of the ramus one may produce a horizontal osteotomy above the lingula, even right away or during the splitting. It has to be treated as such: once the mandible is fixed in the planned position, the proximal fragment has to be fixed to the main fragment with wires or plates. Additional intermaxillary fixation for 3–6 weeks may be indicated.

The Lateral Cortical Plate May Become a Separate Fragment After the Splitting. This free or muscle-pediced fragment must be fixed to the proximal and distal segment of the ascending ramus, followed by 6 weeks intermaxillary fixation. If this is not done, the proximal fragment is free to rotate upwards due to the pull of the temporalis muscle. This may be overlooked as the rotation will not happen as long as the muscles are relaxed. If it is noticed, the proximal segment of the ramus must be fixed to the main segment in the new position. Cutting off the coronoid process facilitates its handling. It will be just the same situation as if a horizontal osteotomy

above the lingula had been planned. If it is not noticed during the operation an ugly situation with an open bite may result. When bony union has resulted in that rotated situation of the proximal segment (Fig. 87i) then it must be sectioned again, repositioned and fixed to the distal main segment. When a gap in the bone results, the necessary bone to bridge the defect is easily obtained by using the lateral cortical plate of the ramus after its splitting or decortication. The bone separator will be very useful for this procedure.

When making the lingual cut to just behind the lingula only, the risk of producing a free fragment is almost zero. But it is possible that the splitting does not work, that the ramus fractures down to the angle without splitting.

Severe Bleeding. It can occur when performing the lingual cortical cut with a rotating burr or with a saw. In most cases the bleeding is caused by damage to the mandibular artery. Bleeding may also come from the internal maxillary artery, although much more rarely. There is no sense in trying to clip the vessel. It is too difficult to find the hole or the stump thereof, and too hazardous for the mandibular nerve. Packing the area will stop the bleeding and permit the operation to be continued. When the splitting has been completed and the fragments are apart, then it might become possible to clip and ligate the artery. If that is not possible or damage to a cavernous haemangioma is the cause of the bleeding, then packing is done with a strip of vaseline gauze, leaving it in situ for 3 weeks. It must be left emerging out of the incision line. The emerging part should be fixed to the soft tissue by a suture.

Damage to the Mandibular Nerve. This is also possible when doing the lingual cortical cut. It is also possible when performing the lateral cortical cut and it is possible to damage it within the ramus during the splitting.

On the lingual aspect, the surgeon may feel he is in the right place above the lingula and in fact he is performing his bone cut below it. There is no question that the nerve and the artery have been damaged or cut. The situation is handled similarly to the description given above for severe bleeding. It should not occur if the semilunar notch has been identified before starting the bone cut.

The nerve and artery may be damaged above the lingula while raising the periosteum without the necessary care. Another possibility I have seen was that the periosteum is perforated in the concavity above the lingula, leaving nerve and vessel on the lateral side of the instrument. They will be cut with the burr in spite of the insertion of the gutter-shaped retractor because they are still close to the lingual cortical plate.

The Lateral Cortical Cut Can Cause Complications. The facial artery or/and the mandibular branch of the facial nerve can be damaged by the saw or the rotating bone

cutting burr when the soft tissues are not well protected by the use of the large gutter-shaped mandibular retractor. The handling of these complications has been mentioned before.

The Contents of the Mandibular Canal Can be Damaged. This can happen when doing the lateral cortical cut as it can run very close to the outer cortical plate, particularly in the premolar and molar areas. Since treatment of this complication is not easy I prefer to avoid it. For that, again I use a 2–3 mm diameter burr, making a broad cut which enables me to recognize when I am approaching the cancellous bone.

During the Splitting of the Ramus Damage to the Mandibular Nerve. Within the ramus it is accepted as “almost normal” by those surgeons who prefer to perform the splitting with thin flexible osteotomes which are supposed to follow the inner side of the lateral cortical plate of the ramus. For the splitting of the ramus itself, no instrument is permitted to pass deeper than 5 mm in between the two cortical plates, as long as we do not have direct vision in between the two segments. This direct vision is best achieved by the use of the bone separator which is inserted into the vertical bone cut and opened gently in stages. This instrument even permits a long surface splitting, far forwards without great risk of damaging the nerve. When the two cortical plates are separated enough to get in between them, the contents of the canal can be visualized and freed under direct vision, when they adhere to, or are actually in, the lateral plate. The bone separator permits the cutting of the posterior border of the ramus with a burr in a long hand-piece when it is reluctant to split, as previously described.

The Splitting Can be Incomplete. Or it may make the lateral cortical plate a separate piece of bone. In the first instance the full splitting has to be accomplished and any minor fragment of the lateral cortical plate handled as a piece of free bone graft. The handling of the second situation has already been mentioned.

When Placing the Screws. The mandibular nerve can be damaged directly by the screws used for osteosynthesis or indirectly by compression osteosynthesis with screws. Careful placing of the screws or plating of the fragments will avoid this. Compression osteosynthesis of the split mandible is contraindicated, in my opinion.

Preservation of the Contents of the Mandibular Canal. It is best done by staying away from it. For that reason also I prefer, in general, the lateral cortical cut to be directed from the second molar area down to the angle area. It does not matter whether it ends a bit in front or behind it.

Some years back, my nephew Joachim Obwegeser was in training at the same clinic where I originally trained

at Graz University and where I had developed the sagittal splitting procedure. I had invited him to come and visit me at my clinic in Zurich for as long as he could, as his chief did not believe in the Le Fort I advancement technique “as all of them end up with a relapse” and in many centres, in cases of sagittal splitting the patients were informed that they would have to accept temporary or permanent anaesthesia of the lower lip. After 3 weeks he said: “Uncle I have now watched you doing ten cases of sagittal splitting and I could not find postoperative numbness of the lower lip in a single case. Now I know that the old saying ‘when two people are doing the same it may still not be the same,’ is right.”

Postoperative Problems

A lot of these exist. However, they can usually be avoided by careful and correct surgery.

Infection. Infection is not a problem when the surgery has been carefully performed. I am convinced of this, even without the use of antibiotics. But when the bone has been burned with the burrs or when large empty spaces are left due to insufficient adaptation of the fragments or when the soft tissues have been badly handled and severely traumatized, massive infection can develop. The treatment has to include complete reopening of the operation field and a thorough washing of it with an antibiotic solution. Two fine vacuum drains are inserted, one above the lingula, the other on the buccal side or in between the segments. An antibiotic solution is instilled every 4 h, through both of them simultaneously, leaving it in for 4–5 min. Naturally, beforehand both tubes have to be rinsed through by applying suction to one of them.

Necrosis of Bone and/or Soft Tissues. I have experienced necrosis of the proximal fragment only once, in a case where the pus contained the same microorganism that was cultivated from a radio-osteomyelitis case which had been operated on in the same operating theatre. I am sure necrosis would not have developed if I had left the periosteum and muscle attached to the lateral aspect of the ramus as we later did, after W. Bell and S. Schendel (1977) had shown the amount of blood supply coming to that lateral cortical plate from the attached muscle.

Dehiscence of the Suture Line. Dehiscence of the suture line will not normally cause a real problem. The wound is rinsed twice daily with antibiotic solution and the open area is either resutured or covered with a vaseline gauze dressing until final healing occurs.

Pseudarthrosis. Pseudarthrosis used not to be too rare a problem. The surgeon's error was that the splitting was too short or a penetrating cut above the lingula had accidentally occurred. I have never experienced a pseudo-

arthrosis after sagittal splitting in my own clinic. It can occur when the neck of the condylar process is cut instead of the lingual cortical cut just above the lingula. Cases had been referred to me in which this had happened bilaterally. The lateral cortical plate of the ramus can be used to bridge the defect.

Non-union. I have observed bone non-union due to a general health problem in some cases. The patients suffered from congenital hypothyroidism. They had received replacement therapy in the form of tablets from early childhood. They had taken their tablets for a long time without being under their physician's control. They had no problems after surgery except for the fact that the split was still movable after 6 weeks intermaxillary fixation, in spite of perfect adaptation of the two fragments. The first case even after twice having an additional 6 weeks fixation, i.e. 18 weeks altogether. When I questioned him carefully, almost like carrying out a psychoanalytical investigation, he confessed that he was taking tablets every day. When he showed them to me I referred him to the department of endocrinology where an iatrogenic hyperthyroidism was diagnosed!

Relapse. Relapse almost always has a local reason, mostly of muscular origin or due to wrong planning. It has been proven that disturbance of oral myofunction is often found in patients with a relapse after surgical correction of antemandibulism (J. Grunert et al. 1990). Accordingly, myofunctional therapy can prevent such a relapse or even cure it (S. Daglio et al. 1993).

Relapse can also have an endocrinological origin. I had experienced this with a female patient with antemandibulism, which I diagnosed as acromegaly. I referred the patient to the endocrinology department and received a negative result. I corrected the antemandibulism by bilateral splitting of the rami, followed by 6 weeks intermaxillary fixation but 3 months after release, the patient had a 100% relapse. The endocrinological result was now found to be positive. As a consequence the adenoma of the pituitary gland was removed. Again, sagittal splitting was used for correction, with 6 weeks intermaxillary fixation. After that the result remained stable. – I have been informed by our endocrinologist that in those days the diagnosis of pituitary gland hyperactivity was not as certain as it is today.

Relapse can also have its origin in unfinished general or hyperactive condylar growth. Scintigraphy of the condyles will be able to answer the question whether or not mandibular growth has ceased.

General Health Complications

These are rare, but they do happen to cause problems. I have heard and read of deaths in connection with the sagittal splitting procedure, either due to severe bleed-

ing which could not be arrested during or after the surgery or due to suffocation. We have, of course, experienced severe bleeding, starting even several days after the surgery, but we have never lost a patient because of bleeding. Once we had a severe airway problem when the patient woke up and was extubated. A gauze swab was found to be the cause; after its removal everything was normal. With today's surgical and instrumental techniques I cannot imagine any lethal consequences after a sagittal splitting procedure.

To complete the discussion on the sagittal splitting procedure, I wish to mention that in any surgical procedures as in anything else, whether it be cars, machines or instruments, there is always the tendency for improvement. If a real improvement or alteration is made, it is generally published.

20.7

Limits of the Sagittal Splitting Procedure

Although the mandible can be made very loose after the splitting, by elevating the musculature and the ligaments with the periosteum all the way around its angle and ascending part, there are limits to the amount of posterior repositioning. After about 20 mm posterior repositioning a stop is experienced, limiting the amount of correction of the occlusion. Apart from the occlusion problem, the aesthetic result is often also not acceptable when the mandible is repositioned so far posteriorly or even more. As long as we did not have the chance to advance the maxilla, for these very severe prognathic cases, osteotomy in the body region seemed the only solution, which I also did not like aesthetically. I then decided to do an angle osteotomy as a variation of the sagittal splitting procedure. I called it primarily prognathism operation procedure Nr. II (H. Obwegeser 1964). Later I termed it the transoral angle osteotomy procedure (see Fig. 86a).

There are, naturally, also limits to advancing the mobilized mandible. When the soft tissues including the periosteum around the mandible are properly elevated it is possible to advance the mandible by up to 20 mm at least and I have done it several times with good results.

Rotating the mandible upwards in the anterior region for closure of a severe open bite reduces the amount of simultaneous elongation in the horizontal direction. However, without simultaneous repositioning of the maxilla, the upwards rotation in the anterior region has quite a tendency to some relapse of the open bite.

Elongation of the ascending ramus in the vertical dimension is often necessary in cases of hemimandibular hypoplasia, independent of its origin. When the periosteum, including the muscle sling, is properly elevated around the angle and the ascending ramus region a vertical elongation of up to 10 mm is easily achievable and even more when the periosteum along the inferior rim of the angle is incised additionally.

The Le Fort I-Type Mobilization Procedure

21.1

Terminology

First a remark on the terminology. All authors who have published on the problem of advancing the retrodisplaced maxilla called it “Le Fort I-osteotomy advancement”. When I started to work on this I also produced real Le Fort I osteotomies. That means the osteotomy line was the same as in a traumatic fracture. What is generally done since I published the mobilization of the maxilla as a standard procedure is not a Le Fort I fracture, as the pterygoid processes are not included. I was wrong to call it a Le Fort I osteotomy. That is the reason why I have changed to the term Le Fort I-type osteotomy, just in order to be correct.

21.2

Historical Background

My teacher, Richard Trauner, and I had a common interest in maxillo-mandibular disharmonies. With his extensive experience in all fields of our specialty, discussing problems with him was very stimulating and rewarding.

We were examining a case of what we then called a pseudoprognathism. It was towards the end of 1952, when Trauner suggested we find a new osteotomy procedure on the mandible with broader contacting raw bone surfaces for the correction of the prognathic mandible. He said, in many cases of so-called prognathism, the cause is the repositioned maxilla, not that the mandible is too far forward. If we could reposition the maxilla anteriorly that would be a tremendous advance in the correction of many facial skeletal anomalies.

It is not intended to report here all historical details on the subject of mobilizing the maxilla, mentioned in the literature. R. Drommer (1986) has published an excellent paper on the history of the Le Fort I-type osteotomy procedure. It makes very interesting and worthwhile reading. I can only repeat what has been published already. However, I do want to mention the very important historical steps of this subject.

The surgical mobilization of the maxilla has a long history. It started with an operation for gaining temporary access to the epipharynx and the base of the skull.

For that purpose, the maxilla was cut horizontally through an extensive horizontal facial incision and also sagittally by dividing the upper lip, which in the German language was first reported by B. Langenbeck (1859) and in the United States by D. Cheever (1867). Temporary mobilization of the maxilla was then used for the same purpose quite frequently, however, using an oral access only. To close an open bite M. Wassmund (1935), reported that in 1927 he had separated the maxilla as a Guérin-type fracture without separating it from the pterygoid processes. He used elastics to rotate the maxilla into occlusion with the mandible. So, that was not a complete mobilization of the maxilla. G. Axhausen reported repeatedly (1934, 1936, 1939) successful anterior repositioning of the maxilla in post-trauma patients as well as in cleft cases by the use of elastics after complete osteotomy of the maxilla, including separation of the pterygoid processes from the maxilla. K. Schuchardt (1942) after he had detached the pterygoid processes in a second operation, used weight traction to reposition the maxilla in a post-traumatic war case. He stated that this procedure would have a large application in cleft cases, but in such cases it will probably never come into use.

When, in 1975, I was invited by R. Dingmann to Ann Arbor as a guest speaker to a postgraduate course on cleft lip and palate, to explain my philosophy on the correction of secondary deformities in cleft cases, I presented cases for which I had done the maxillary advancement in one, or two or three pieces for unilateral and bilateral cleft cases and I presented cases of Le Fort III advancement osteotomy and Le Fort III and simultaneously additional Le Fort I corrections (H. Obwegeser 1969). In the discussion, Dingmann congratulated me on the results and said that we Europeans were far ahead in the correction of these anomalies. I replied that Dingmann himself had published in 1951 on Le Fort I and Le Fort III osteotomies in posttraumatic cases. He said, did I? He had forgotten.

J.M. Converse and H. Shapiro (1952) and I. Cupar (1954) used an almost circular incision in the vestibulum for the horizontal osteotomy and in addition they raised the whole palatal covering in performing the transpalatal and retromolar osteotomies. As for mobilizing the maxilla completely, the nasal mucosa must

also be reflected, that procedure does not seem to leave enough blood supply to the maxilla, when it is moved forward for quite a distance. It did not come into general usage either.

When I trained with Sir Harold Gillies in 1951/52 I had the privilege of assisting him in quite a number of cleft cases in which he did a Le Fort I-type osteotomy for correcting the collapsed segments of the maxilla and their vertical deficiency. Through the help of N.L. Rowe the segments were fixed in the new position by cast cap splints with a connecting bar, attached to a headcap of plaster of Paris. The gaps between the laterally and downwardly rotated maxillary segments were filled with cancellous bone chips. These bone transplants were covered by the mobilized vestibular mucosa only, but were otherwise in open communication with the antral and nasal cavities. They all healed well. However, I never saw him mobilize a maxillary segment or the whole maxilla completely for repositioning it further anteriorly. Also in the publication of H.G. Gillies and N. L. Rowe (1954) all illustrated cases were handled in the same way as "greenstick fractures", as also did H.G. Gillies and R. Millard in 1957. The same is true for the cases shown in the publications of P. Cernea et al. (1955) and J. Levisnac (1958).

In the German literature W. Widmaier (1960) reported that his chief, Eduard Schmid, had corrected the retrodisplaced maxilla in several cleft cases by performing a Guérin-type fracture on the two halves of the maxilla. A horizontal vestibular incision was used to expose the anterior surface of the maxilla and the osteotomy was performed with an osteotome. Occasionally, E. Schmid tunnelled the vestibular mucosa to secure the blood supply to the maxilla in cases in which the very scarred palatal mucosa had to be "detached". Separating the junction between the pterygoid plates and the maxilla was accomplished with an osteotome via the maxillary sinus. Sometimes it was additionally necessary to detach the pterygoid processes through a small incision in the palate. By rotational movements of the osteotome or by a gentle blow with the hammer, the alveolar process of the maxilla was made sufficiently mobile. That is the description of E. Schmid's procedure by his co-worker W. Widmaier. Nothing is mentioned about additional mobilization or advancement of the maxillary segments. The results of the cases shown in that publication are the result of lateral rotation of the maxillary segments only, similar to those of H.G. Gillies and R. Millard (1957). The cases published by W. Widmaier also do not permit the conclusion that the maxillary segments had been advanced. Regrettably, there is no publication in the literature up to that date and later, by E. Schmid himself, in which he reports on that subject, showing cases of real maxillary advancement. I worked for him for several months in 1954 and before, and I never saw him advancing a maxilla. I have, however, learned a lot

from him. I consider him to be one of my very important teachers. He was very innovative.

It seems astonishing that a clear working procedure had never been developed for advancement of the retrodisplaced maxilla, in spite of the fact that many surgeons had seen the need for it and had tried to do it. g. Axhausen (1934, 1936a,b, 1939) seems to have been the only surgeon who has frequently shown results of the repositioning of the maxilla after its mobilization, however, by the use of elastics. For some reason his technique also did not come into use.

When I lectured on the full mobilization of the maxilla at a postgraduate course at Walter Reed Hospital in Washington D.C. in June 1966, not one of the over 500 participating surgeons seemed to have ever done or seen it done.

In 1969 I visited my good friend Ralph Millard in Miami, for almost a week. A few days before, I had just presented the previously mentioned paper on "Surgical Correction of Deformities of the Jaws in Adult Cleft Cases" at the First International Conference on Cleft Lip and Palate, at Houston. My colleague and friend from Erlangen, Gerhard Steinhard, was also there for some days. Ralph asked us to present a paper to his staff. G. Steinhard's paper (1969) on spontaneous regeneration of large mandibular defects after subperiosteal resection in animals and patients was very impressive. I presented the same paper that I had given at the Houston meeting. Almost all of his staff and trainees did not agree with our philosophy regarding the need for dual qualification for the work we were doing.

Then we were asked to give our opinion on some trauma cases which had been treated by them. Our philosophy of facial fracture treatment was different to theirs. One case presented to us was treated 1 year before, but he still had a retrodisplaced maxilla, including one zygoma. The horizontal occlusal gap was about 10 mm. Our suggestions for correcting that situation were invited. The answer was simple: bring forward surgically what the trauma had displaced. They wanted me to demonstrate it to them as they would not be able to do it. As mobilizing the zygoma is quite difficult without proper instruments I suggested I would do the maxillary osteotomy and bring it into proper occlusion and they should then deal with the depressed zygoma if this was still requested by the patient. They agreed. Days went by. At the end of that week I had to leave at noon for the airport. Nothing had happened. On the last day of my visit at 10.30 a.m. I was watching Ralph Millard performing his technique of face lifting when someone asked me to do that case straight away. I asked them to prepare the necessary wire splints on the teeth and take some cancellous bone from the iliac crest for grafting behind the tuberosities and in both canine fossae. They worked hard in two teams, one in the mouth and the other on the hip. At 11.00 A.M. I was asked to come to the

operating room again. The wire splints on the upper and lower teeth were not finished and the piece of bone taken from the iliac crest was waved proudly in the air by an ambitious young surgeon. But it was just enough for one side! He had to reopen the donor site on the hip and take another piece of bone and the team working in the patient's mouth also still needed another 20 min to finish the splint. It was almost 11.30 a.m. when I was able to step in and start, with everything else except a bone cutting machine and instruments to which I was accustomed. It was a bit of a "rough operation". Within 15 min, the maxilla had become so mobile that I could overcorrect it with a pair of tweezers. I did the inter-maxillary fixation and asked them to do the bone grafting for me as I had to leave for the airport. The young surgeons were obviously pleased and thanked me. Dr. Ben Hur from Israel, one of the trainees, apologized that he had attacked us so vehemently, disagreeing with the necessity for dual qualification for maxillofacial surgery. He said, he was now convinced.

One year later I was there again for a short visit. The case was shown to me. He had a perfect occlusion and mouth opening. But he had a fistula in the back of his upper vestibulum discharging a little pus. I was asked why was this the case. With a probe I could follow the fistula behind the tuberosity area. No cause was possible in my opinion other than an overlooked piece of gauze. The following day it was found and removed.

Summarizing, I dare to state that around 1960, neither in Europe nor in the USA, did there exist a clear procedure for mobilizing the maxilla in such a way that it could be placed wherever needed. But there was definitely a need for a standard procedure which enabled the safe mobilization of the maxilla for the correction of its anomalies of whatever origin. It was a challenge to find also a procedure for correcting most of the abnormal positions and forms of the maxilla, after the sagittal splitting procedure on the mandibular rami and the

chin sliding procedure had introduced the possibility to correct most of the common mandibular anomalies. Following my principle "*A necessity can neither be forbidden nor be eliminated by ignoring it*" (Principle No. 37), I started to work on it.

21.3

Steps in the Development of the Le Fort I-Type Standard Operation Technique

In the "late fifties", when I was already working at Zürich University, I had to deal with post-traumatic retromaxillism in several cases. I was of the opinion that what the trauma can push backwards I must be able to bring forwards. In order to secure as much blood supply as possible to the maxilla, I wanted to leave the vestibular mucosa attached to it. From vertical incisions in the midline and behind the canine and in the molar regions, I raised the mucoperiosteum, sectioned the anterior surface of the maxilla with Lindemann burs (Fig. 88a) and detached the nasal septum after I had elevated the nasal mucosa. For that I had the forked-type nasal septum osteotome made. I also cut the pterygoid plates horizontally, using an osteotome, as the trauma had also fractured them. For the mobilization itself, I used a pair of Rowe's disimpaction forceps. When it seemed to be loose enough, I pulled the maxilla with wires into the planned occlusion. After 4 weeks inter-maxillary fixation a certain amount of relapse occurred again. I did not know why. In the next case I did the same and again got some relapse. It then occurred to me that it was due to the backward pull of the pterygoid musculature. In the following case I separated the tuberosity area from the pterygoid plates and again performed a proper mobilization. I obtained a good occlusal result in that case (Fig. 88b).

After these simple Le Fort I-type post-traumatic cases, we had an open bite case which, according to the occlusion, was also post-traumatic but not as a 1- piece fracture. It was my first case involving cutting the maxilla into two unequal pieces for repositioning them upwards in the posterior regions (Fig. 88c).

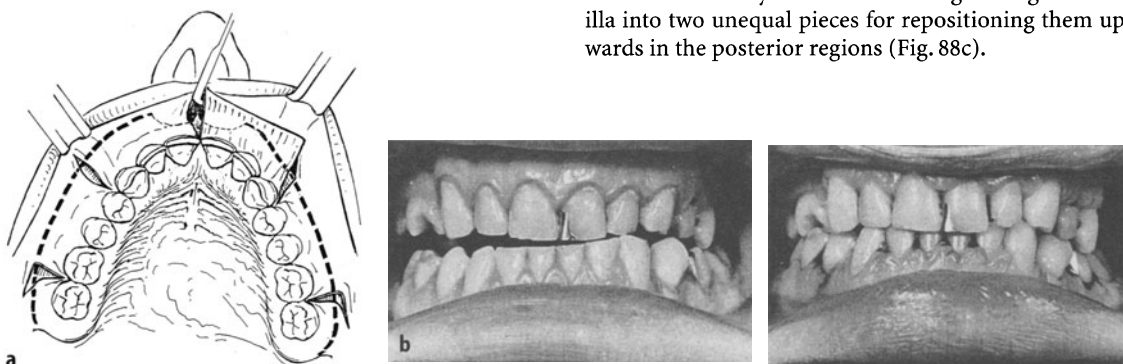


Fig. 88a, b. The development of the Le Fort I-type mobilization. **a** Vertical vestibular mucosal flaps for securing blood supply to the mobilized maxilla. **b** First successful case of mobilization of the maxilla in a late post-traumatic situation, for correction of its retrodisplacement (from H. Obwegeser 1962)

That experience I then used for the correction of a collapsed and retrodisplaced maxilla in an unilateral cleft case. Somehow it was very difficult to mobilize the maxilla. I was glad that there was a vestibular blood supply as the palatal mucosa looked badly damaged by the use of the disimpaction forceps. After I had reopened the cleft, I could swing the fragments nicely laterally. But the mobilization and the advancement was not really complete. After 6 weeks intermaxillary fixation the result was not bad, but also not perfect (Fig. 88d).

The number of cases seeking improvement of their dish-face deformity due to retromaxillism increased. Particularly the cleft cases, with collapse of two or even three segments of the severely retrodisplaced maxilla,

were a challenge. With the vertical vestibular mucosal flaps securing the blood supply to the maxillary segments in unilateral cleft cases, it was still difficult to mobilize the maxillary segments well enough after the cleft had been reopened. I did not dare to cut the vestibular mucosa horizontally. I was frightened to interfere too much with the palatal blood supply when advancing the maxillary segments by 10 mm and more. I had watched Sir Harold Gillies using horizontal vestibular incisions when I trained with him in 1951/52. But he only rotated the segments laterally and I never saw him advancing these segments. He fixed them in the new position with the help of cast cap splints which were attached via a vertical bar to a head cap. Once he had stabilized the ro-

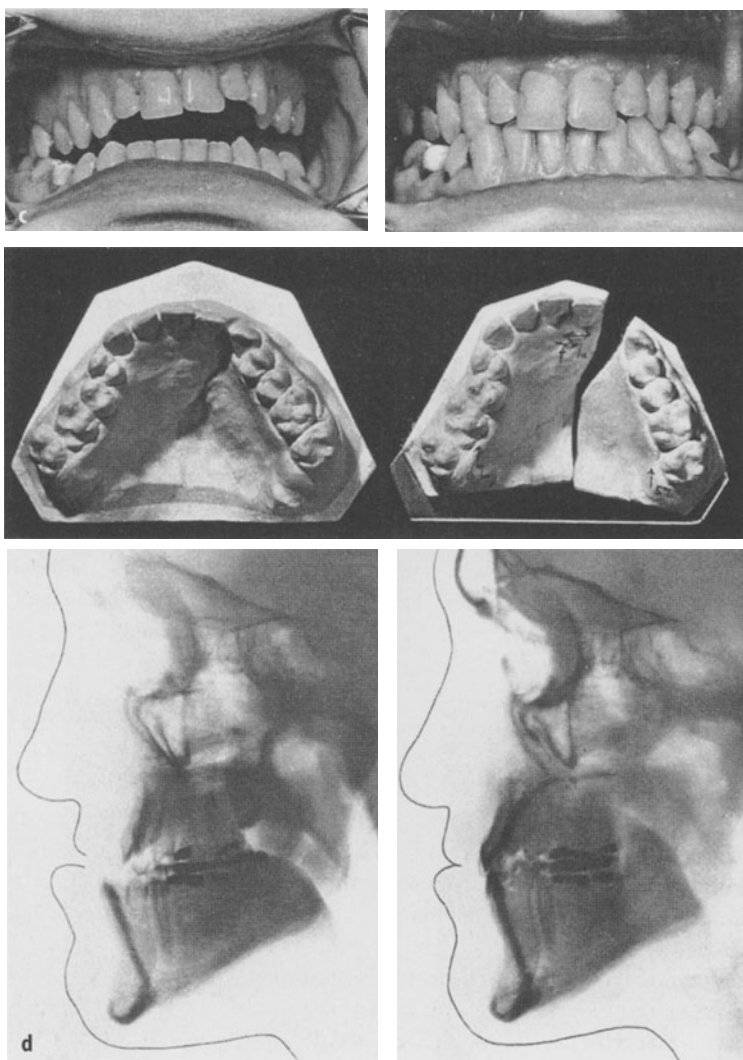


Fig. 88c, d. The development of the Le Fort I-type mobilization. **c** First case of mobilization and sectioning the maxilla into two pieces for closure of a post-traumatic open bite (from H. Obwegeser 1964). **d** First unilateral cleft case in which the maxilla was sectioned into two pieces and advanced for correction of severe retromaxillism together with collapse of the dental arch (from H. Obwegeser 1965)

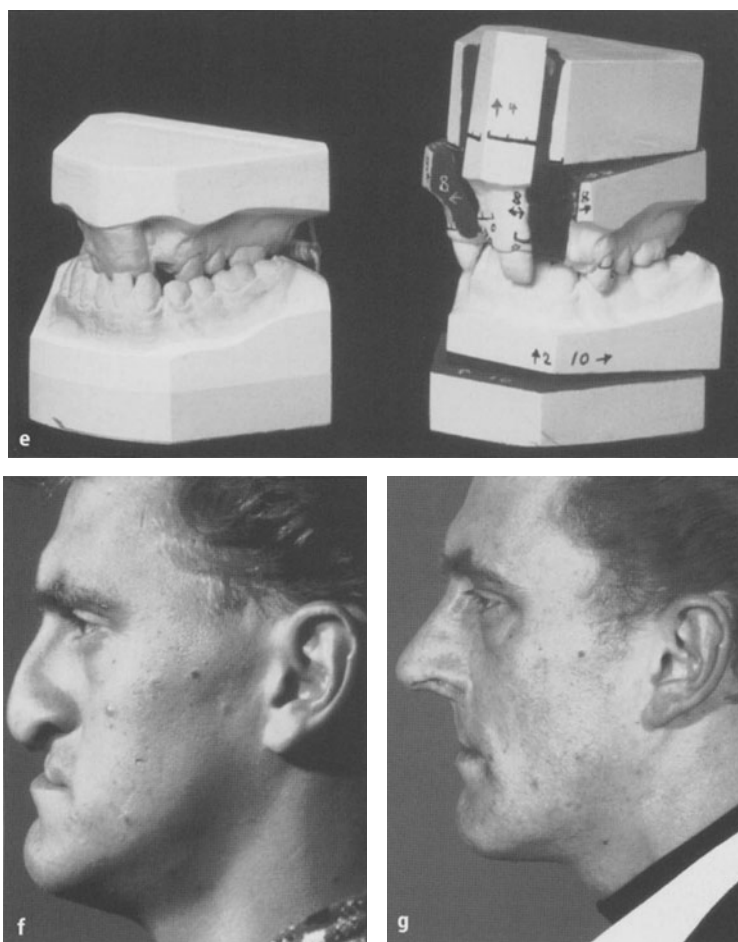


Fig. 88e-g. The development of the Le Fort I-type mobilization. **e-g** Very severe retromaxillism in a bilateral cleft deformity case, corrected by swinging the two main segments laterally and the front segment forwards and correcting the sagittal discrepancy with a mandibular set back operation. Additionally, the lip was reoperated on and simultaneously the columella was elongated by the use of Millard's forked flap technique (from H. Obwegeser 1966)

tated segments he placed cancellous bone grafts on the steps in the canine fossa, leaving them in wide open communication with the maxillary sinuses. He then mobilized the vestibular mucosa and covered the bone grafts with it on the vestibular side only. The horizontal discrepancy he corrected by repositioning the mandible. I also did this in very severe dish-face deformity cases which were due to a collapse of all three maxillary segments in bilateral clefts (Fig. 88e-g) if I was unable to advance them far enough. The vertical vestibular mucosal flaps limited the amount of advancement of the maxillary segments.

21.3.1 The All-Decisive Case

Then the all-decisive case turned up. It forced me to change my approach. The patient was a young man, the

18-year-old son of a famous Swiss family, who was referred by the chief of plastic surgery of the University Hospital. In a car accident, 6 weeks earlier, he had lost three upper incisors and suffered an open bite, due to an untreated telescoping Le Fort I-fracture in two pieces. So far, I had always used vertical vestibular incisions leaving mucosal bridging flaps attached to the maxilla. In this post-traumatic case the maxilla was not only split into equal halves but also retrodisplaced and telescoped into the nasal and maxillary sinuses, producing not only a retromaxillism condition but also a severe open bite in the anterior region (Fig. 89a). Additionally there were several communications between the oral cavity and the nose (Fig. 89b). The three upper incisors were missing. In the vestibulum on both sides, a scar ran as far back as the zygomatic crest. The model operation proved that a perfect occlusion in the cheek teeth regions could be achieved when the maxilla was mobi-

lized and cut into two pieces (Fig. 89c). Because of the scars in the vestibulum, there was no chance of leaving vestibular mucosal flaps attached to the maxilla to ensure a safe blood supply. Because of that vestibular scar I made an incision from one zygomatic crest, through the oro-nasal fistulae, over to the other zygomatic crest. I removed the scar tissue in the former fracture lines. As the two halves were telescoped into the maxillary sinuses I had to free them there and perform osteotomies from the canine fossae over the zygomatic crests posteriorly. I carefully observed the bleeding of the fragments during the vestibular osteotomies which were accomplished by the use of osteotomes and thin fissure-type burs. It remained satisfactory. Then I raised the nasal mucosa all the way back along the nasal floor and the lower part of the septum. The bleeding of the maxilla still remained satisfactory. Next, I detached the septum from the hard palate with the forked nasal septum osteotome and cut the lateral nasal walls below the inferior nasal conchae. The blood supply still remained satisfactory. Next, I separated the maxilla from the pterygoid plates using a slightly curved, rather strong osteotome. For mobilizing the maxilla I did not dare to use the disimpaction forceps for fear of damaging the palatal blood supply as there were some post-traumatic scars. Instead I pushed the maxilla in the front region inferiorly. This was later called the downfracture technique when I did it routinely in all the subsequent cases and demonstrated it to many visitors. With the strong, slightly curved osteotome I tried to pull the maxilla gently forwards on each side.

Naturally, I was frightened to interfere with the blood supply as in the literature all surgeons who had split the

maxilla in two halves and rotated them inferiorly and laterally had stated that the descending palatine artery must remain intact for the necessary blood supply to each half of the maxilla. It worked and the blood supply became poor but recovered after a while. I had to separate the two halves of the maxilla in the palate also, in order to be able to place them independently into proper occlusion (Fig. 89d). Direct bone wires were used to secure the two halves of the maxilla in the correct position. The blood supply to the two halves of the maxilla remained satisfactory. The oro-nasal communications could be closed after some mobilization of the vestibular mucosa and also the palatal opening between the two halves of the maxilla could be closed simultaneously. The occlusion achieved (Fig. 89e) was equivalent to that before the accident.

This case demonstrated that the maxilla receives an adequate blood supply from the palate even when it is mobilized. It does not need any blood supply from the vestibular, nasal or antral mucosa. A large advance in the technique of mobilizing the maxilla was achieved. This was the actual "birthday" of today's Le Fort I-type mobilization procedure. It was in 1964.

However, knowledge concerning the development of the Le Fort I-type osteotomy procedure was not finished. I still had to learn to deal with the most severely retrodisplaced, collapsed maxillae of unilateral or bilateral cleft patients. I had also to learn how to overcome the tendency to relapse in these cases. The majority of cases badly needing an advancement of the maxilla were cases of secondary cleft deformity. In addition to the advancement they also needed an increase in maxillary height as their cleft maxillary segments were not

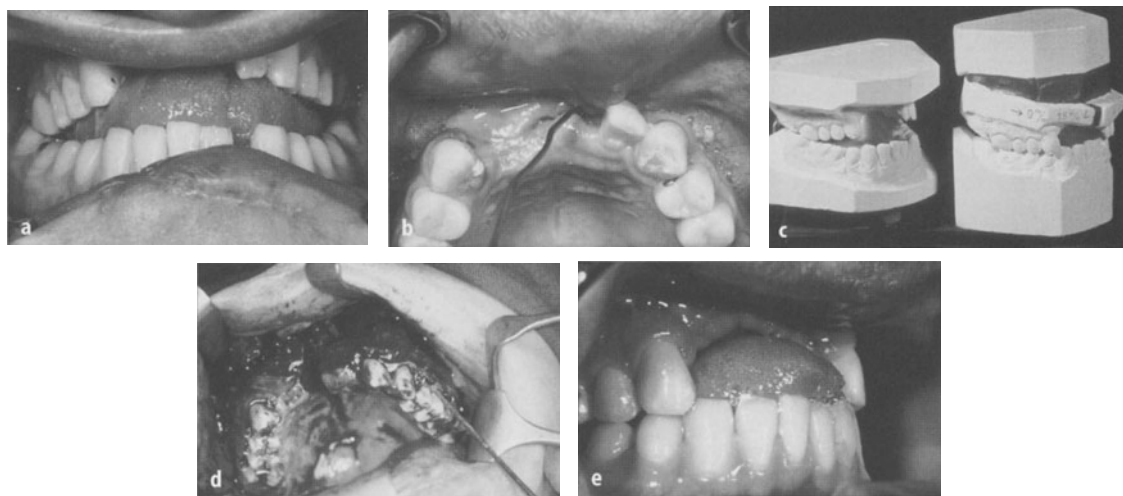


Fig. 89a–e. First case of advancement of the maxilla in two segments in spite of horizontal vestibular incisions (from H. Obwegeser 1967). **a** Severe open bite in the front region due to a post-traumatic telescoping retromaxillism in two segments. **b** Oro-nasal communication and vestibular scarring on both sides. **c** Model operation. **d** The completely mobilized two halves of the maxilla. **e** Occlusion achieved

only collapsed and too far back but they also had a severe lack of downward growth. In most of them their mandible was in a normal position but their prognathic appearance differed very much, depending on the degree of retromaxillism.

21.3.2

The Final Step: Bone Grafts to the Osteotomy Defects

Then a very severe unilateral cleft deformity case was referred to me. The maxilla was slightly collapsed and far back while the mandible was in the normal position for a straightforward profile type (Fig. 90a, b). Occlusion was present in the molar regions only (Fig. 90c). The profile planning indicated that the mandible should remain untouched. The model planning showed that a forward movement of the maxilla was required by moving both segments independently, 15 mm on one side

and 13 mm on the other side (Fig. 90d). Because of the rather large steps in the canine fossa regions I planned to use bone grafts to bridge these defects and also between the pterygoid processes and the tuberosities. As the right infraorbital region was even flatter than the left I intended to place there also a piece of bone graft to improve the contour. The operation plan was to bring the two maxillary segments so far forward that the mandible could remain untouched (Fig. 90e).

The mucosal covering of the hard palate looked reasonable, but there was a rather long fistula between the small and the large segments. I used the circular vestibular incision learned from the previously mentioned post-traumatic case. The osteotomies in the canine fossae and in the lateral nasal walls were performed using a Lindemann bur after the nasal mucosa had been elevated and protected. The separation of the maxilla from the pterygoid process was done as in the previous cases and so was the detachment of the nasal septum from its crest.

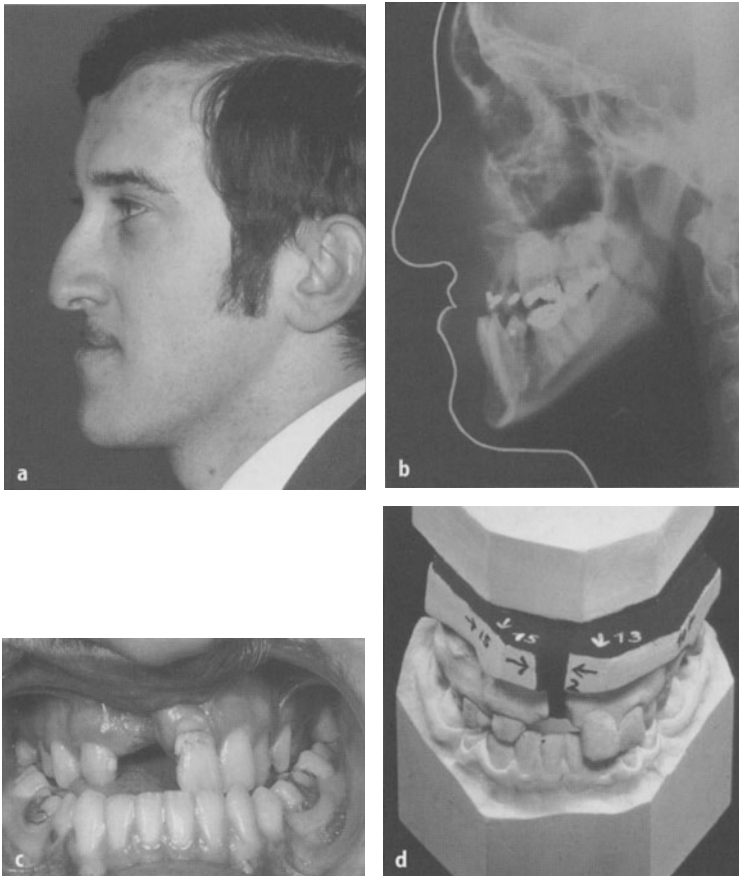


Fig. 90a–d. First case of correction of retromaxillism of 15 mm in a unilateral cleft deformity (from H. Obwegeser 1969). **a, b** Patient's presurgical profile view and lateral cephalogram disclosing severe retromaxillism with dish-face deformity and typical cleft humpy nose. **c** Presurgically there was occlusion in the molar regions only. **d** The model operation showed that the two maxillary segments had to be moved forwards independently

There was enormous resistance to the mobilization of the maxilla in spite of the strong force I used. Then I opened the cleft again, rotated the two halves laterally. Now I could feel with a finger where the scars were which had prevented the advancement of the maxilla. I cut them carefully. Finally, I got both segments absolutely loose and far enough forwards with their blood supply still satisfactory. The distance between the tuberosity and the pterygoid process measured 15 mm on the side of the lesser segment as determined in the model operation. Due to the lateral rotation of the two halves there was also a large step in the canine fossa and in addition a bit of a vertical defect, as both segments also had to be rotated downwards. It was obvious that these bone gaps had to be filled with bone. I used blocks of cancellous bone from the iliac crest, 15 mm long behind the small segment and 13 mm long behind the major segment. For

the horizontal and vertical defects in the canine fossa, the bone pieces were cut accordingly and fixed with wires. After mobilization of the vestibular mucosa the bone grafts could be covered on the vestibular side. But they stayed in wide open communication with the maxillary sinus and the nasal cavity. The postoperative course was uneventful. Aesthetically and occlusally the result was very satisfying (Fig. 90f,g). The unilateral cleft remained open. It had to be closed in a separate operation later, using local tissue and bone grafting.

With this case, the technique for advancing the maxilla had become a safe standard procedure (H. Obwegeser 1962, 1964, 1965, 1966a, 1967a, b). The bone grafting to the open maxillary sinus I had seen when I trained with Sir Harold Gillies in 1951/1952. He swung the two segments laterally producing a greenstick fracture at the pterigo-maxillary junction as did E. Schmid accord-

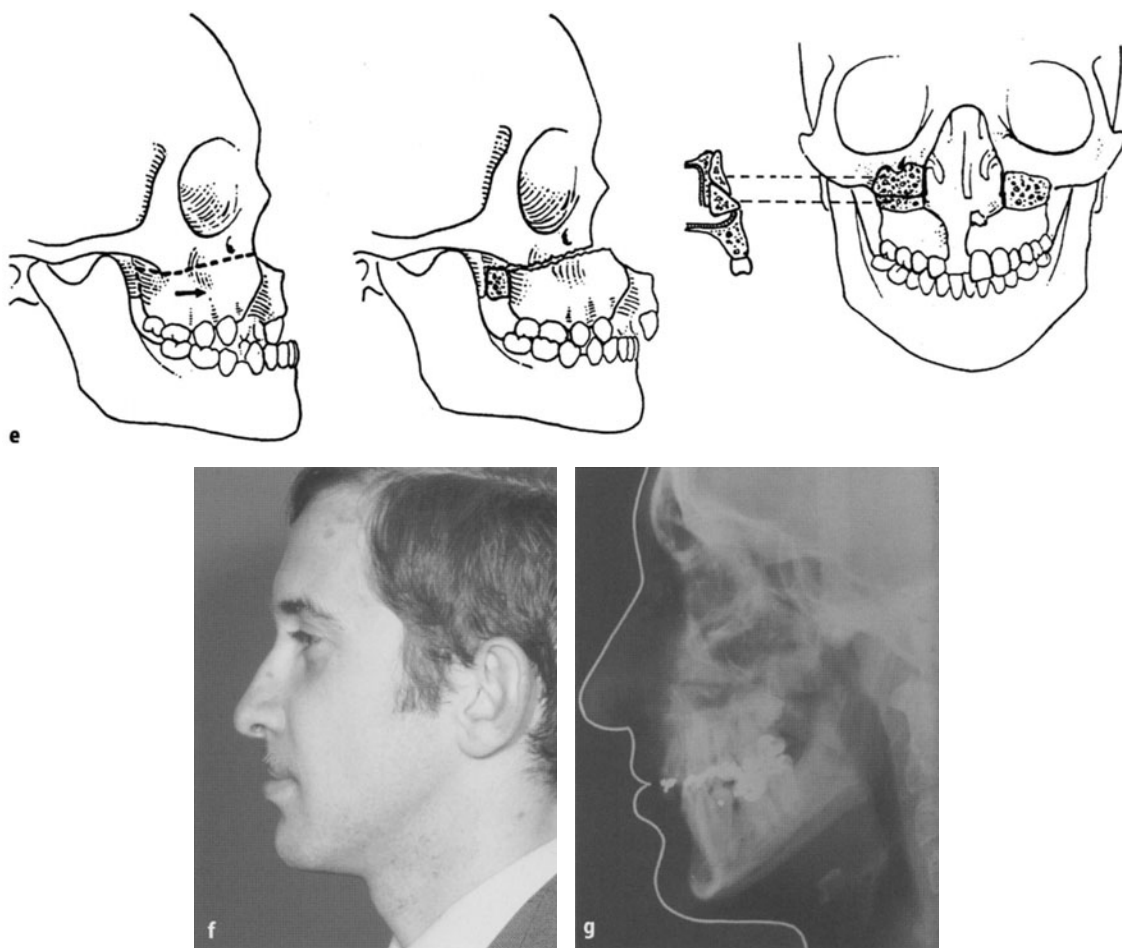


Fig. 90e–g. First case of correction of retromaxillism of 15 mm in a unilateral cleft deformity (from H. Obwegeser 1969). **e** Diagrammatic illustration of the planned operation. **f, g** Patient's profile view and lateral cephalogram 1 year after surgery. The humpy nose was automatically corrected just by the advancement of the maxilla. The large bone block behind the maxilla is easily detectable on the lateral cephalogram

ing to W. Widmaier (1960). Later, there were also cleft and non cleft cases in which I had to move the maxilla forwards by up to 20 mm.

21.3.3

Closing the Defect of a Missing Tooth

In almost all cases of cleft maxillary advancement, the cleft had to be reopened to permit the achievement of an acceptable occlusion. The reopened cleft had to be closed again in a second operation. Later, I closed the alveolar cleft in unilateral cases simultaneously by advancing the small segment with its teeth into direct

tooth contact with the main segment. This meant that the canine tooth was brought into contact with the first incisor of the major fragment or even its first bicuspid with the second incisor (Fig. 91a–f).

21.3.4

Deep Frozen Bank Bone in Maxillary Advancement

In many cases in which I had to advance the maxilla between 5–10 mm only, I routinely used deep-frozen human cadaver cancellous bone with excellent results. I placed it behind the advanced maxilla and at the step in

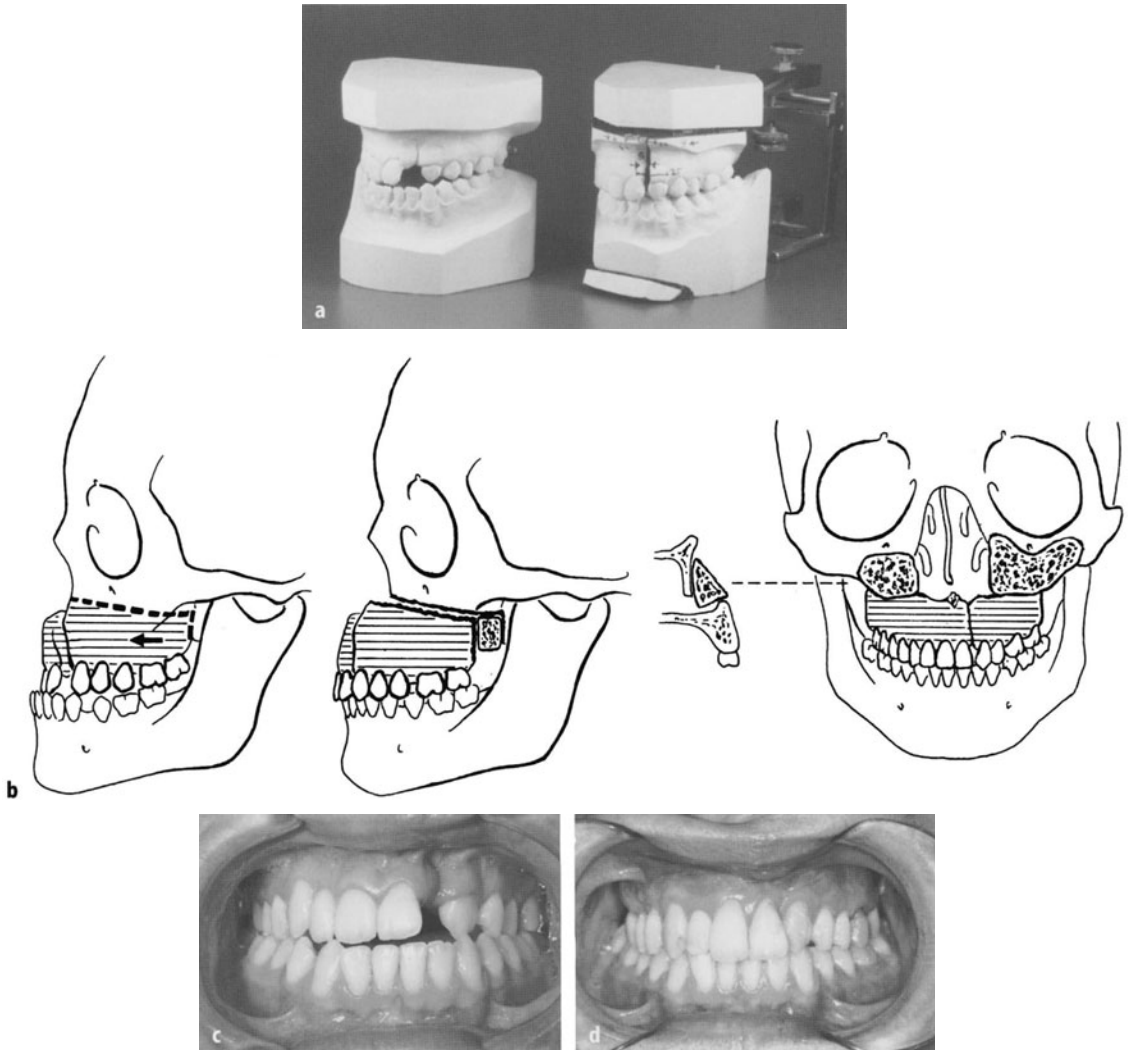


Fig. 91a–d. Simultaneous advancement of the two segments of a cleft maxilla and closing the gap of a missing second incisor (from Obwegeser 1988). **a** Model operation indicating that a very acceptable occlusion can be achieved when both segments of the maxilla are advanced and rotated together. **b** Diagrammatic illustration of planned procedure. **c, d** Occlusion before and 1 1/2 years after surgery



Fig. 91e, f. Simultaneous advancement of the two segments of a cleft maxilla and closing the gap of a missing second incisor (from Obwegeser 1988). **e, f** Patient's pre- and postsurgical appearance

the canine fossa. In cases requiring more than 10 mm advancement, it did not produce bony union. In those cases I then used the patient's own cancellous bone

wedged in between the tuberosity area and the pterygoid plates and the canine fossa step (H. Obwegeser 1969b,c).

21.4

First Case of Simultaneous Advancement of the Maxilla and Repositioning of the Mandible

Then a case turned up with such a severe horizontal discrepancy between the two dental arches and the middle and lower facial thirds that neither with a maxillary advancement alone nor with a mandibular set back operation alone could the facial disfigurement be corrected properly as well as the planned occlusion achieved. This situation was found in both non-cleft and cleft cases. Formerly I had tried to improve the aesthetic as well as the occlusal situation by repositioning the mandible only. The achieved occlusion was acceptable but not so the aesthetic result. I also had experienced that there is some limitation to pushing the mandible backwards by the sagittal splitting procedure. It had become unavoidable that in these cases the maxilla had to be advanced as well as the mandible repositioned. I decided to do this in one operation. It was performed on 5 September 1969.

The main indication in the first patient to have repositioning of the mandible and advancement of the maxilla in one operation was his severe micro- and retromaxillism, well recognizable in the appearance (Fig. 92a) as well as in the lateral cephalogram (Fig. 92c). The occlusion was that of a severe bilateral class III relationship (Fig. 92b). As a consequence of the severe mi-

cro-maxillism, there was a large distance between the upper and lower teeth when the patient's mandible was in its resting position (see Fig. 92c).

The occlusion result would always be the same, whether the mandible or the maxilla alone were moved or both simultaneously (Fig. 92d). The necessary amount of vertical height increase of the maxilla could be measured on the lateral cephalogram, taken in the resting position of the mandible (see Fig. 92c). In order to find out the amount of repositioning of the maxilla and the mandible in the sagittal dimension that would be ideal for the final result, the desired profile line was drawn on a transparent sheet over the existing profile line. The sagittal difference of the two gave an idea of the necessary amount of sagittal movement of the two jaws. I planned to make the final decision on the operating table.

The necessary amount of maxillary advancement and the additional necessity to increase the vertical height of that very small maxilla made it clear that I had to add bone in the canine fossa as well as behind the maxilla. The bone grafts for the canine fossa had to be shaped like a step in order to secure the necessary

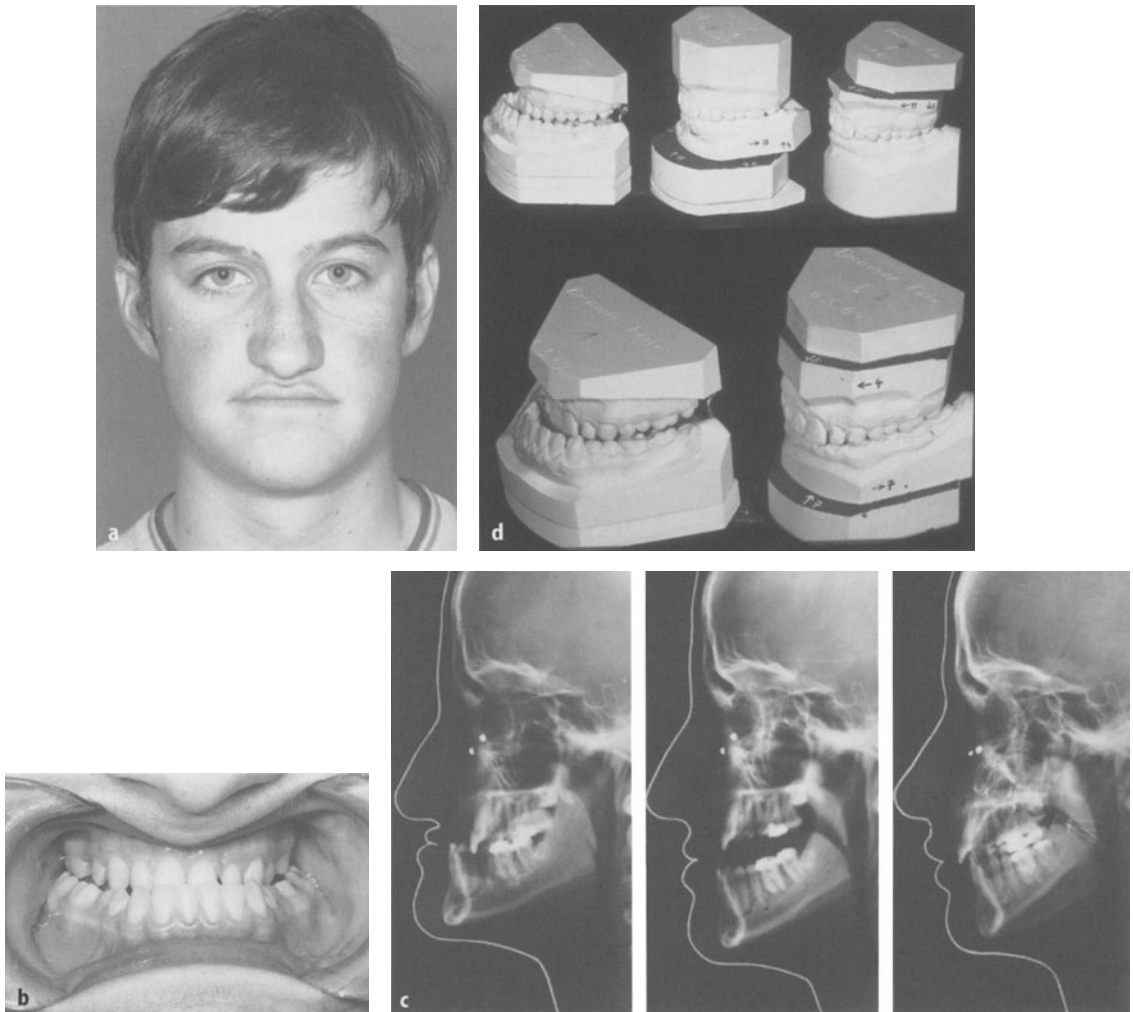


Fig. 92a–d. First case of simultaneous advancement of the maxilla and repositioning of the mandible for correction of a very severe sagittal discrepancy (from H. Obwegeser 1970). **a, b** Patient's appearance and occlusion before surgery. **c** Lateral cephalograms, before surgery in the closed and mandibular resting position and 1 1/2 years after surgery. **d** Model operation demonstrating that the occlusion will always be the same whether the maxilla or the mandible alone or both are repositioned

amount of vertical height increase of the maxilla as well as to reduce the flatness in the canine fossa region (Fig. 92e).

A continuous loop wiring splint was fixed to the teeth to secure the planned occlusion for 6 weeks with intermaxillary fixation. During surgery there were no real problems. When both jaws were completely mobile I fixed them together. Now I could move the jaws as one firm block. I fixed this provisionally in position according to my imagination and checked the patient's appearance. As I found it satisfactory I then completed the operation.

After 6 weeks both jaws were firm. The occlusion was as planned. There was not only full bony union, but due

to the bone grafting to the canine fossa, a welcome contouring of the face resulted. There was some over-contouring at the beginning but after 1 year the facial contour was just right (Fig. 92f) and so was the occlusion (Fig. 92g).

When it had become a routine procedure in my hands to reposition simultaneously the whole maxilla as well as the mandible (H. Obwegeser 1970) this was not only the solution for the correction of the very severe sagittal discrepancy of the two jaws but it also opened the door for modern corrective surgery of the lower half of the facial skeleton. Nowadays, many surgeons rarely do one jaw only, most do both at the same time, even if it is for 2–3 mm repositioning only.

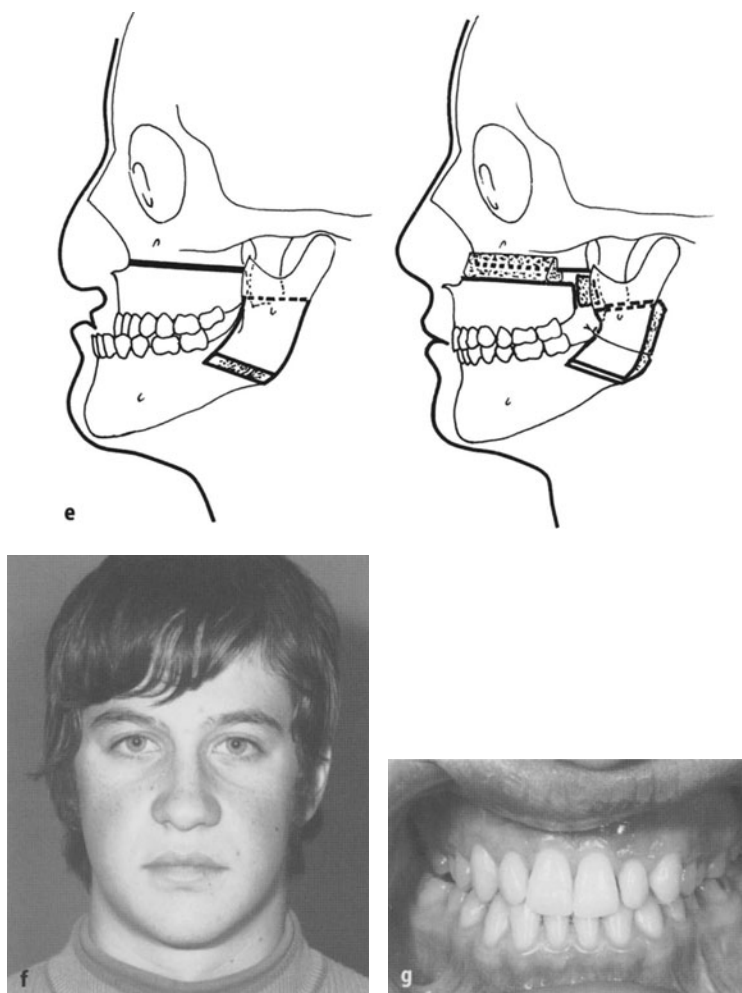


Fig. 92e–g. First case of simultaneous advancement of the maxilla and repositioning of the mandible for correction of a very severe sagittal discrepancy (from H. Obwegeser 1970). **e** Schematic illustration of first case of simultaneous repositioning of the mandible and advancement of the maxilla with additional increase in its height. **f, g** Patient's front view and occlusion 1 year after surgery

When I lectured on the “Surgical correction of Deformities of the Jaws in Adult Cleft Cases”, at the First International Conference on Cleft Lip and Palate at Houston in 1969, I showed various types and degrees of secondary cleft deformities and their correction, including my first cleft case of simultaneous repositioning of the maxilla, even in sections, and the mandible. I was then asked whether I was such a bad primary cleft surgeon that I

was producing such poor late results. However, I had seen such and also worse cleft deformities all over the world. They very much depended on the technique used and the radicalness of the primary cleft surgeon. But with this procedure of repositioning the maxilla and the mandible, as a whole or in sections, it was now possible to produce a normal facial skeletal framework out of a very distorted one (H. Obwegeser et al. 1978, 1985a).

21.5 Modifications and Further Progress

Just as modifications of the sagittal splitting procedure had been advocated, there were several modifications suggested for the Le Fort I-type osteotomy, mainly in

the antral wall area. Some osteotomies with steps, others descending or ascending or even curved.

21.5.1

Kufner's Extended Osteotomy of the Maxilla

In my opinion, the only one which brought additional improvement was J. Kufner's suggestion (1971) to include a part of the inferior orbital rim and zygoma (Fig. 93a). Quite a high percentage of cases, in particular the cleft cases, require a contour improvement higher up in the face than just at the base of the maxilla. Instead of performing that extended Le Fort I-type osteotomy, I often camouflaged the flatness of the paranasal and infraorbital-zygoma area by inserting a piece of cancellous bone at the step in the canine fossa area. Or, I im-

planted bilaterally an L-shaped piece of lyophilized cartilage paranasally (see Fig. 56l), if there were no occlusal indications to advance the maxilla.

Because of the good results obtained in the correction of maxillo-mandibular disharmonies, other cases were referred which needed more than just repositioning of the maxilla and the mandible. There were cases which required an advancement of the full middle third of the face, some even including the forehead. Sir Harold Gillies had told me that he had tried to perform a Le Fort III advancement and warned me not to do it, as it was too hazardous. Nevertheless, I tried, again first in post-traumatic cases. When I related this in summer

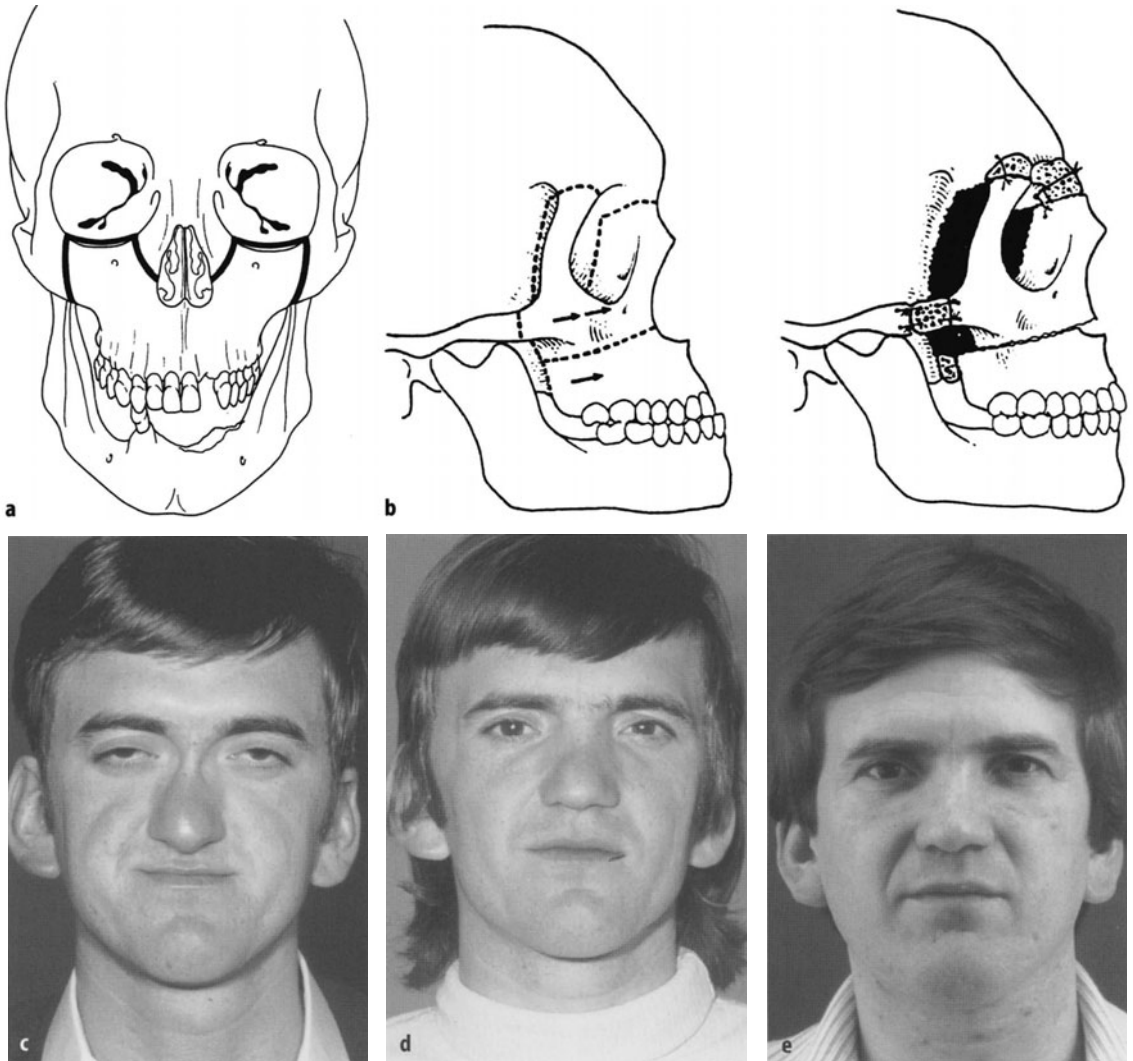


Fig. 93a–e Extension of the Le Fort I mobilization. **a** Kufner's procedure includes the infraorbital rims. **b** Diagrammatic illustration of simultaneous Le Fort I and III mobilization for correction of severe dish-face deformity (from H. Obwegeser 1969). The procedure applied in an 18-year-old patient with severe midface hypoplasia and syndactylism of the upper and lower extremities, of unknown origin (radiologically no M. Apert). **c** front view before, **d** 3 years and **e** 11 years after surgery

1967 to Dr. J. Dautrey, a French visitor, he informed me that his chief, Dr. Paul Tessier in Paris, has produced a technique to advance the middle third of the face and also to correct hypertelorism. I met Dr. Paul Tessier in September 1967 in Rome at the International Plastic Surgery Congress. We became very good friends and I learned a lot from him, to the benefit of many patients. I consider him as my last real teacher.

21.5.2

The Lower Two Thirds of the Facial Skeleton

After P. Tessier (1967, 1968; P. Tessier et al. 1967) had published his Le Fort III operation procedure and the correction of hypertelorism, the ultimate road towards the non-limited correction of cranio-maxillo-facial skeletal anomalies was opened. It was then obvious that in craniosynostosis cases, as well as in some ordinary cleft cases, in which the whole middle third of the face required advancement, the maxilla could be separated from the upper half of the middle third in order to reposition it, if necessary in two segments, according to the needs of the occlusion, and the upper half according to the needs of the orbits (H. Obwegeser 1969b,c, 1973b) (Fig. 93b–e).

While P. Tessier's Le Fort III and his hypertelorism procedure with their variations opened up the possibility of correcting any skeletal anomaly of the upper half of the facial skeleton, so did the sagittal splitting of the rami procedure and the Le Fort I mobilization together with the alveolar segmental corrective surgery (H. Obwegeser 1968) and the trans-oral chin correction enable us to correct even the most severe occlusal and skeletal configuration anomaly of the lower half of the facial skeleton.

When I had operated upon little Antonio with his two complete noses and a wide medial facial cleft the little 11-year-old boy had acquired quite a reasonably pleasing result (Fig. 94a,b) in spite of the fact that I had to remove the reconstruction of the defects of the lateral orbital walls due to massive infection. That infection also necessitated the neurosurgeon taking away the huge bone flap of the craniotomy (H. Obwegeser et al. 1978). When the boy had finally completed his general skeletal growth, a grotesque facial and occlusal picture had developed (Fig. 94c–e) due to my inexperience of what damage too early surgery can do to the middle third of the face. To correct this very severe facial skeletal and occlusal anomaly, I had formulated the following operation plan (Fig. 94f): Advancing the maxilla by an extended bilateral Kufner Le Fort I-type mobilization and additionally separating the upper parts from the severely retroplaced maxilla for correction of the lack of vertical height. This should correct the severe dish-face deformity, apart from the missing nasal bridge. Simultaneously, a mandibular set back operation along with retropositioning of the lower anterior alveolar segment should create an acceptable intermaxillary relationship (Fig. 94g). For the reconstruction of the missing nasal bridge I planned to use an L-shaped piece of cartilage, as I had learned from E. Schmid (1956), simultaneously with the necessary elongation of the columella by a caterpillar flap (Fig. 94h). The nose turned out well (Fig. 94i).

Regrettably, in those days we still used infraorbital incisions to approach the orbits for the correction of the extremely severe hypertelorism. Otherwise the extremely severe facial and occlusal deformities were very satisfactorily corrected by altering the various parts of the lower two-thirds of the facial skeleton (Fig. 94j–l).

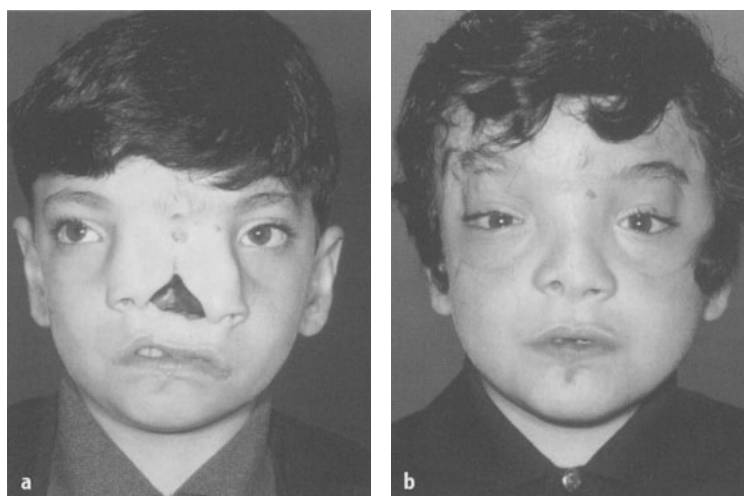


Fig. 94a, b. Very severe facial skeletal and occlusal anomaly after correction in childhood of a midfacial duplication and extreme hypertelorism (from H. Obwegeser et al. 1978), **a, b** 11-year-old boy with facial duplication before and after correction

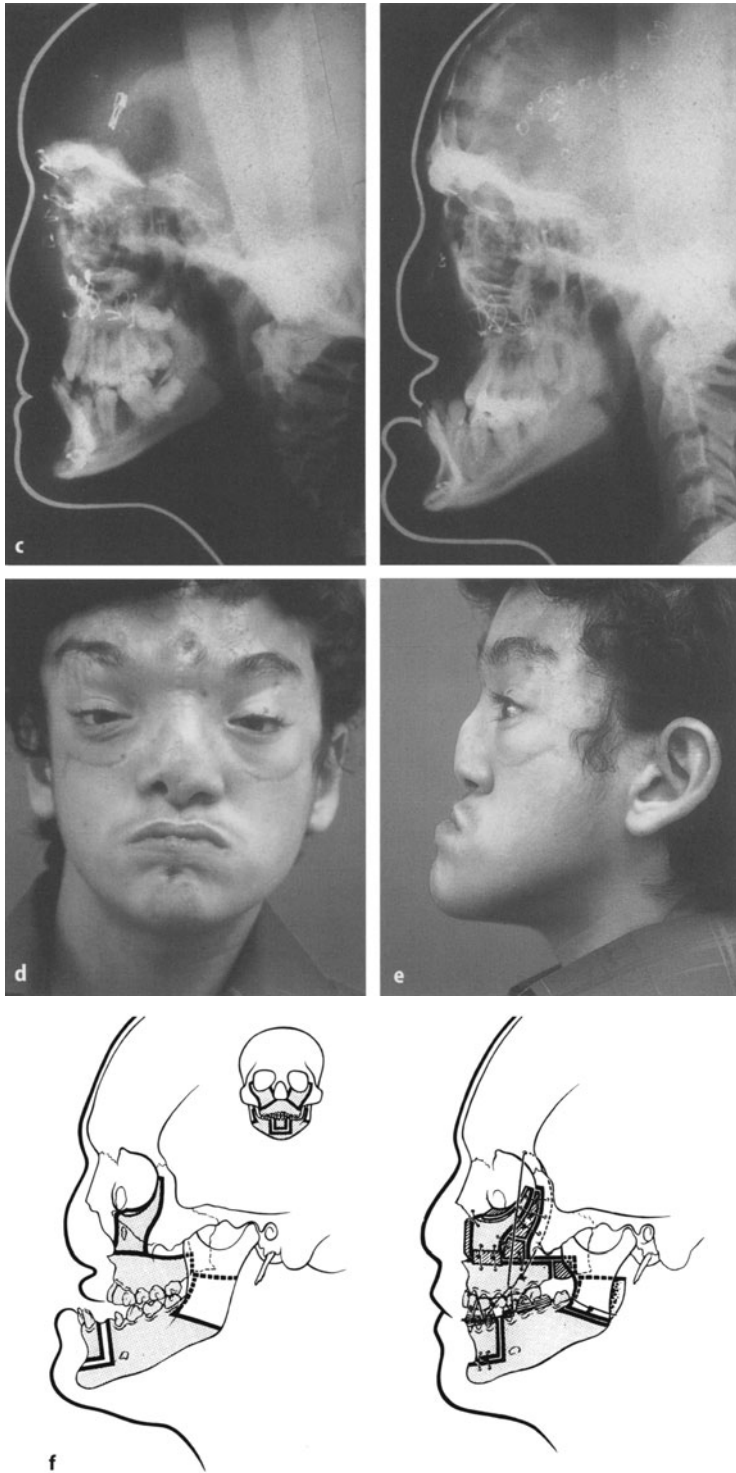


Fig. 94c–f. Very severe facial skeletal and occlusal anomaly after correction in childhood of a midfacial duplication and extreme hypertelorism (from H. Obwegeser et al. 1978). **c** Lateral cephalograms showing severe lack of growth of the patient's middle third within 6 years of correction. **d, e** Patient's front and profile views with extreme facial deformity at age 18. **f** Schematic illustration of planned correction of the patient's facial skeletal deformity

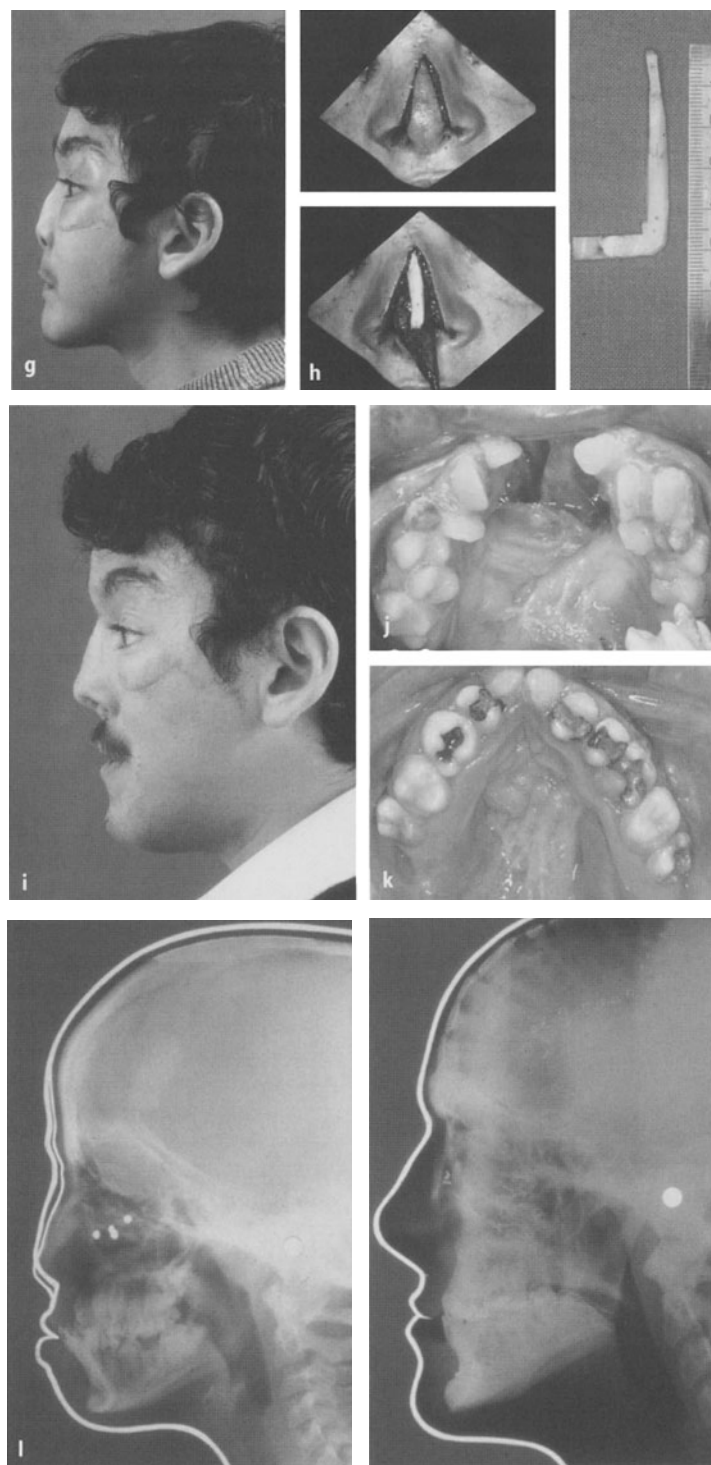


Fig. 94g-l. Very severe facial skeletal and occlusal anomaly after correction in childhood of a midfacial duplication and extreme hyper-telorism (from H. Obwegeser et al. 1978). **g** Profile view of the result of the correction. **h** Reconstruction of the missing columella by a caterpillar flap and the missing nasal bridge by an L-shaped cartilage. **i** Patient's appearance in profile view 1 year after the last surgery. **j, k** Situation of the maxilla before surgery and the final result. **l** Lateral cephalograms before first correction and 1 year after the last surgery

21.5.3 The Whole Facial Skeleton

Using present day surgical procedures, it is possible to correct in one operating session, very severe cranio-maxillofacial skeletal anomalies such as those seen in Apert's syndrome deformities. The skeletal anomalies in such cases can include all three thirds of the facial skeleton. It is worthwhile demonstrating such a case for discussion of some important points (Fig. 95).

The upper and middle thirds of the facial skeleton are usually much too far posterior in these cases. Apart from that, in most cases teleorbitism is present as well as a micro- and retromaxillism (Fig. 95a,b). The mandible is not directly involved in the craniostenosis. So, it can be of normal shape, or it can also show signs of some type of malformation, including a misregulation of growth anomaly.

The planning of the corrective surgery is done as usual, on tracings of the lateral cephalogram, to ascertain the necessary amount of forward movement of the forehead and the middle third of the face (Fig. 95c,d) and on model operations for the correction of the occlusion (Fig. 95e).

The craniotomy is planned in such a manner that not only allows the forehead to be moved anteriorly by the necessary amount, but the design also allows good fixation of the advanced and rotated orbits. Then the mid-facial complex is mobilized, the orbits together with the maxilla. This procedure, without separating the maxilla, is called the monobloc advancement of the upper two-thirds of the facial skeleton (F. Ortiz-Monasterio 1978).

In addition to that monobloc advancement, many craniostenosis cases require not only an independent

amount of advancement of the orbits and the maxilla as shown in Fig. 93b, but also rotation of the orbits and reduction of their distance apart (Fig. 95f,g). For that the maxilla is then separated from the orbital cones and the desired occlusion with the mandible is confirmed.

In these cases the maxilla is often not only too far back but also so small that presurgical orthodontics requires extraction of several teeth on each side in order to be able to place the remaining teeth in correct angulation to the small maxillary base. The maxilla may be so small that on each side only four or five teeth can be left. If the teeth are orthodontically protruded, instead of achieving the necessary space by extractions, the orthodontist limits the surgeon's possibility of advancing the maxilla far enough forward (Fig. 95h,i).

Next, the surplus width of the nasal framework, together with the underlying ethmoid cells is reduced and then the orbits are rotated together and fixed in their new position. The osteotomies within the orbital cones should have been made far back in order to be able to rotate the orbital contents, also when the orbits are brought together. Next the maxilla is fixed to the mandible in the planned occlusion. Then the surgeon has to decide whether he can fix the maxilla directly to the united orbital cones or whether he has to interpose a bone graft on each side for correction of an existing vertical deficiency. After the maxilla is fixed to the upper half of the middle third of the facial skeleton, the intermaxillary fixation is released again and a canthopexy is done on both sides.

If the position or shape of the mandible must also be corrected, that will now be done according to the new position of the maxilla. Before closing the incision in the upper vestibular sulcus, the desired nasal correction is performed or a bridge augmentation is carried out.

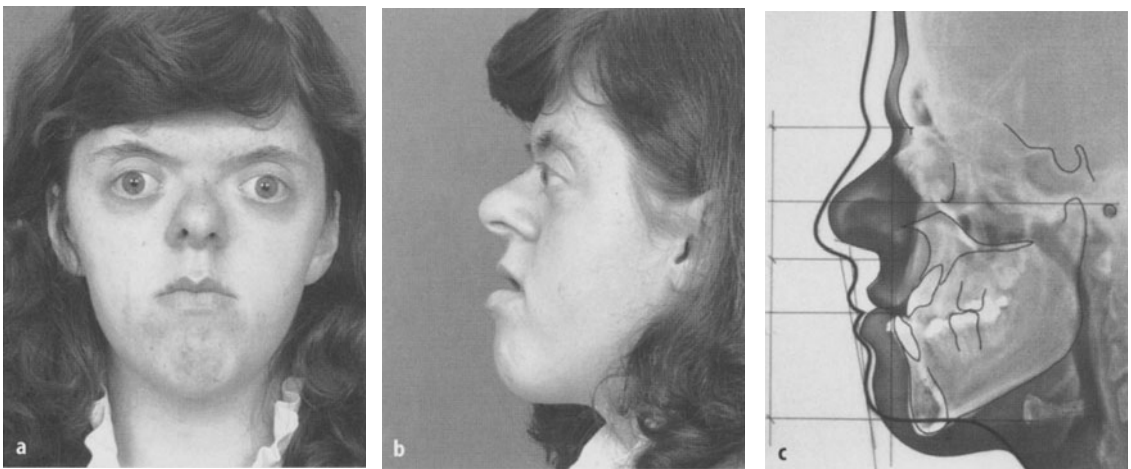


Fig. 95a–c. Correction of the whole facial skeleton in a case of Apert's syndrome deformity. **a, b** Patient's front and profile view disclosing the very severe retroposition of the upper two facial thirds, accompanied by teleorbitism and additional hyper- and retrogenia. **c** The profile planning

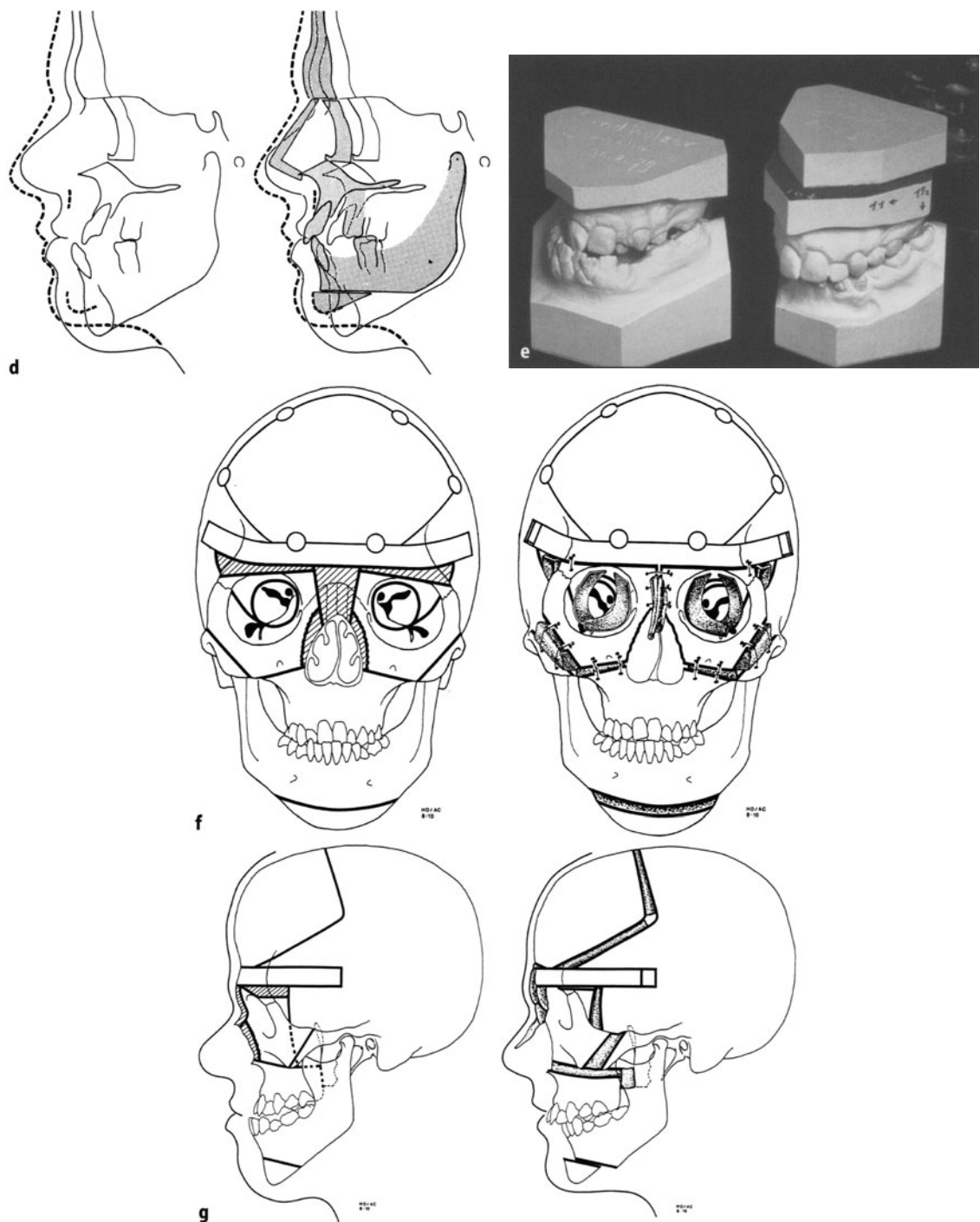


Fig. 95d-g. Correction of the whole facial skeleton in a case of Apert's syndrome deformity. **d** Evaluation of the amount of the necessary sagittal movement of the various facial skeletal parts. The upper half of the middle third has to be advanced by 20 mm. **e** The model operation indicates that for occlusal reasons the maxilla requires advancement by 11 mm. **f, g** Diagrammatic illustration of planned skeletal correction, monobloc advancement of the upper two-thirds of the face plus correction of the malposition of the orbits and independent advancement of the maxilla and correction of the retro- and hypergenia

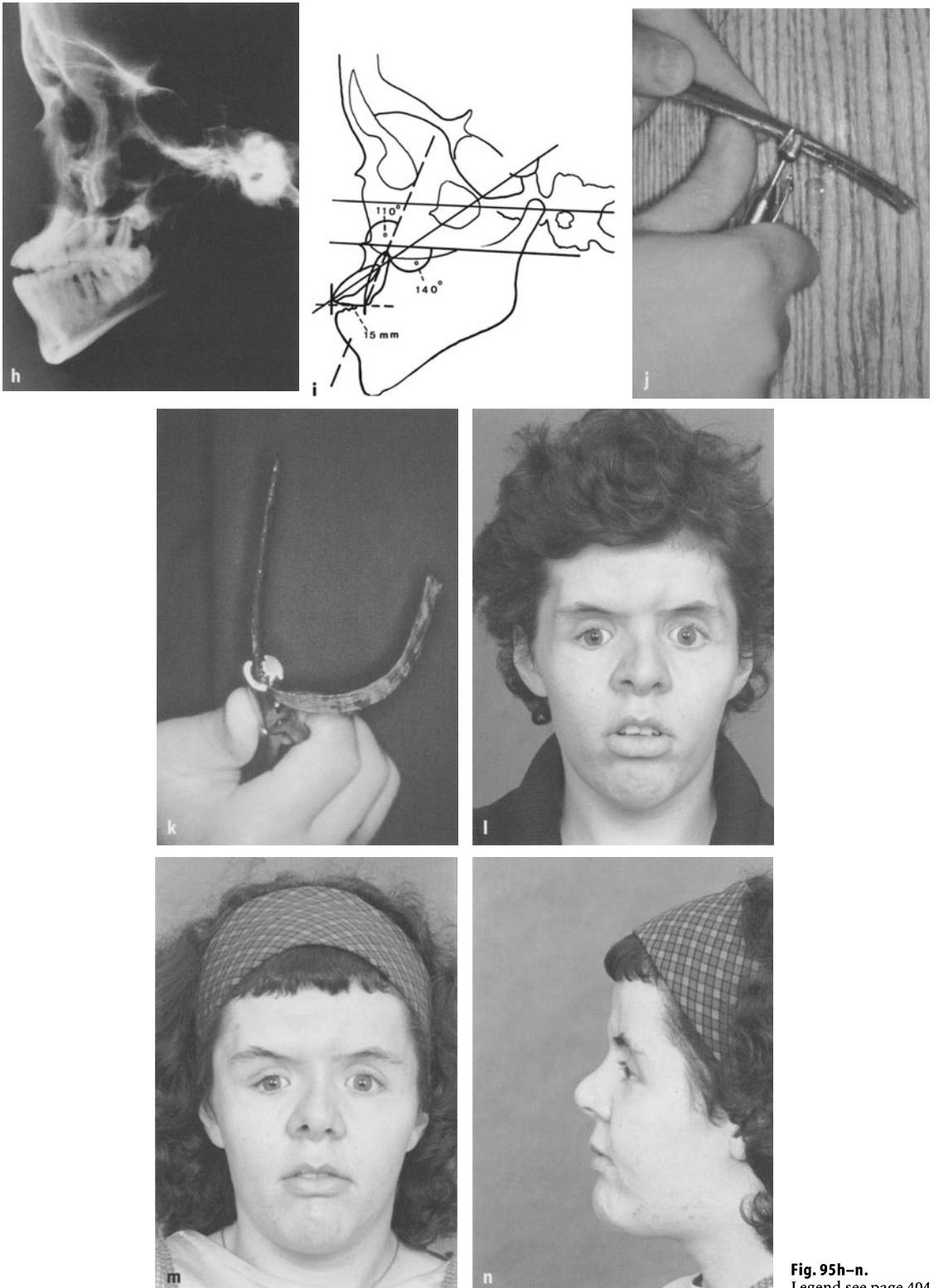


Fig. 95h–n.
Legend see page 404

The defects in the lateral and medial orbital walls as well as in the floor and roof of the orbital cones are reconstructed. Cranial bone can easily be obtained from the bone flap which was removed for gaining the necessary access to the anterior base of the skull. I am very fond of using split rib grafts. First, I take off most of the cortical covering of the rib with an acrylic bur and then I take off the rib edges with the same bur (Fig. 95j). Next, the rib is split with thin osteotomes or a strong knife. Finally the rib is bent as required by the use of bone-contouring forceps (Fig. 95k). The decorticated half of a rib can be formed and bent better than any other piece of bone. With this technique I have been able to reconstruct a skull defect the size of almost half of the skull (see Fig. 94c and 94l).

Due to the amount of orbital advancement, a bilateral enophthalmos may result, as well as an unsightly de-

pression in both temporal areas (Fig. 95l). These secondary defects can be reconstructed some months after the main operation. For the correction of the enophthalmos, I will again mobilize the orbital contents completely and place behind the equator of the eyeball enough very thin slices of lyophilized cartilage. They must be cut with the dermatome so thinly that the globe compresses them against the walls of the socket. It requires some experience to know what is the correct amount to produce a good result. If too much cartilage has been placed resulting in overcorrection, the surplus can easily be removed later as the cartilage slices remain as if they had just been placed. I also use the same material to fill the depression in the temporal areas. So, finally in two operations a very pleasing result can be achieved, from the aesthetic point of view as well as functionally (Fig. 95m, n).

21.6

My Final Version of the Le Fort I-Type Mobilization

Mobilization of the maxilla is now most probably more often performed than repositioning of the mandible. The technique may differ from one surgeon to another, although not very much. It has only changed slightly in my hands since 1970. In a straightforward case, the mobilization itself should not take much longer than 10–15 min.

Approaching the Maxilla

With the patient under naso-tracheal intubation anaesthesia, either the blood pressure is lowered, or a vasoconstrictor is injected into the vestibular incision and tuberosity areas. I perform the vestibular incision horizontally through the mucosa and then, in one cut, vertically to the alveolar process through the periosteum, about 5 mm above the attached mucosa, using a scalpel. Some prefer electrosurgery. The incision goes all the way around from one zygomatic crest to that on the other side. Where it ends, on top of the zygomatic crest, I direct the incision obliquely dorsally upwards for some millimetres to achieve enough access to the tuberosity area without elongating the incision by tearing it with the retractor. Then the mucoperiosteum is elevated up to the infraorbital nerve. I want to know where it is. In

the front it is raised above the nasal spine. On top of the zygomatic crest I incise the periosteum vertically for 5–8 mm. Next I make marks with a bur on the zygomatic crest and in the canine fossa. These will enable me to measure the amount of forward or backward movement of the maxilla. If the maxilla has to be raised I mark the amount of excision necessary on the antral wall. Next, the edge of the pyriform aperture is freed and the mucoperiosteal covering of the floor of the nose is raised on both sides along the whole length of the hard palate and on the lateral walls of the nasal cavities up to the inferior turbinates. On the medial aspect along the nasal crest of the palatine bones, elevating the muco-periosteum without tearing may present some difficulties. Care must be taken to prevent this from happening especially when approaching the juncture of the nasal crest of the palatine bones with the septum. Maintaining an intact muco-periosteum is even more important when a deviated septum is being corrected at the same time.

Separating the Maxilla

The periosteum is then carefully raised behind the zygomatic crest and into the pterygo-maxillary junction.

Figure is on page 403

Fig. 95h–n. Correction of the whole facial skeleton in a case of Apers syndrome deformity. **h** Lateral cephalogram of another case of Apert's syndrome before presurgical orthodontics, the severe protrusion of the upper teeth makes it impossible for the surgeon to advance the maxilla by the amount necessary to produce a good facial appearance. **i** When the upper front teeth have been repositioned into proper angulation to the base of the maxilla by orthodontic means, additional advancement of the maxilla by 15 mm will be possible. **j** Preparation of a rib graft for splitting and bending. **k** Forming the decorticated half of a rib with the bone-contouring forceps for the reconstruction of the defects in the orbital walls. **l** After the 1-stage correction of the facial skeletal anomaly in accordance with the plan, a normal facial skeleton and intermaxillary relationship resulted, but with a rather severe bilateral enophthalmos and a concavity (depression) in the temporal regions due to the 20 mm advancement of the orbits. **m, n** Patient's final appearance 1 year after correction of the enophthalmos and the temporal depressions by subperiosteal implantation of thin slices of lyophilized cartilage

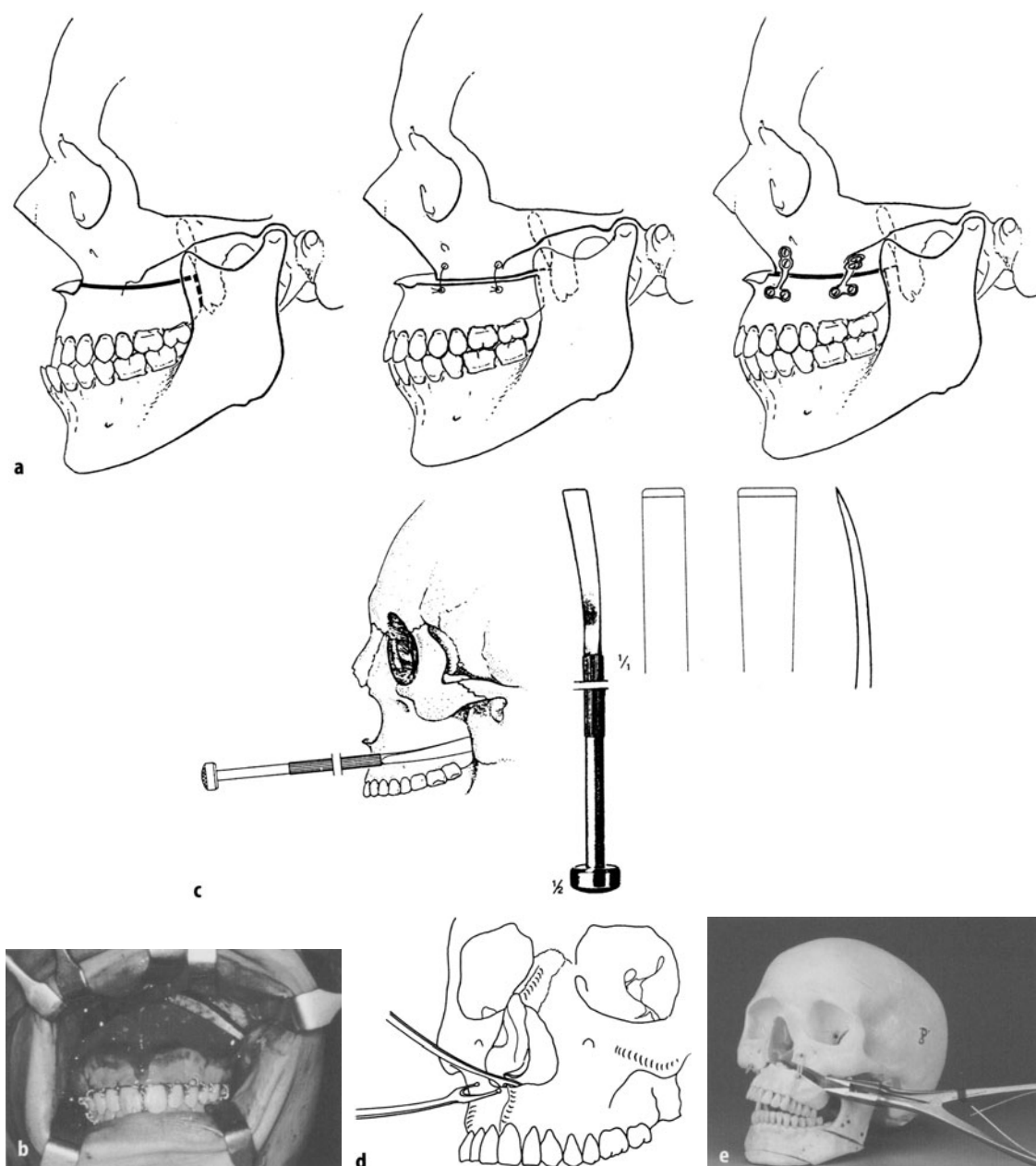


Fig. 96a–e. My final version of the Le Fort I-type mobilization and its fixation with wires or plates and screws. **a** Schematic drawing of the osteotomy lines for the Le Fort I-type mobilization. **b** Strips of bone removed with the saw for correction a maxillary long face. **c** Thin, slightly curved osteotome for separating the junction between the maxilla and the pterygoid process. **d** The nasal septum osteotome in action. **e** The posterior downfracture of the maxilla with the bone separator

If it is torn, Pichat's fat pad will come through the opening in the periosteum into the operation field. That annoying situation is best handled by excising what has come through and then covering the opening with a flat piece of gauze which is held by the retractor, which is now to be inserted. I prefer the upwardly bent retractor inserted at the junction between the maxilla and the pterygoid process.

I still use the same horizontal osteotomy for separating the maxilla as I did at the beginning when I started to use this procedure in non-trauma cases, occasionally varying it a bit from behind up, or downwards to the front region (Fig. 96a).

For the osteotomy of the walls of the maxillary sinus I like to use thin disposable blades in the reciprocating saw to cut the antral wall from posteriorly forwards including the lateral nasal wall, where a periosteal elevator prevents the mucosa being cut. Others prefer a bur and use an osteotome for the lateral nasal wall. I hate bone fragments in that area because of possible obstruction of the lacrimal duct and also possible postoperative nasal problems.

There are other reasons why I prefer to use a thin disposable blade in the saw to perform my osteotomy of the maxilla. With it, the loss of bone is negligible compared with the use of burs or even osteotomes. Also, I can direct the cut according to the planning, upwards or downwards; in addition I can remove exactly that strip and shape of bone which I need in my correction of a maxillary long face (Fig. 96b).

Next the pterygo-maxillary junction is separated using a very thin, slightly curved and slightly flexible osteotome (Fig. 96c). The posterior antral wall is cut with the saw with that osteotome in place.

The nasal septum is now detached from the hard palate using the forked septal osteotome (Fig. 96d). For safety reasons, the posterior pharyngeal wall may be protected from the forked septal osteotome being driven into it by packing the epipharynx with a large strip of gauze. The latter should not be forgotten! For that reason a black thread is fixed to it, emerging through the mouth. This packing is removed before the end of the operation and definitely before intermaxillary fixation.

The Actual Mobilization of the Maxilla

When all bony connections of the maxilla to the higher bones are cut, now comes the mobilization. For quite some time now I have ceased commencing with the frontal down fracture. I learned from my nephew J. Obwegeser to bring the maxilla inferiorly first in the posterior part. To do this, I insert the narrower bone separator from the zygomatic crest further posteriorly and open it gently. The maxilla comes down easily (Fig. 96e). Then the front part is lowered, again with the bone separator inserted at the nasal aperture rim. This is the reverse of my previous procedure. This ensures that the maxilla is really freed from the pterygoid processes.

To advance the maxilla and complete the mobilization further, I no longer use the so-called maxillary mobilizer type elevator (Fig. 96f). The necessary force is not always that easy to control and I am also not in favour of using Tessier's maxilla mobilizers also because of the possibility of applying too much uncontrolled force. I had an instrument made by the Medicon Company which I called a "maxillary advancer" (see Fig. 102d). With one leg resting on the zygoma, the other

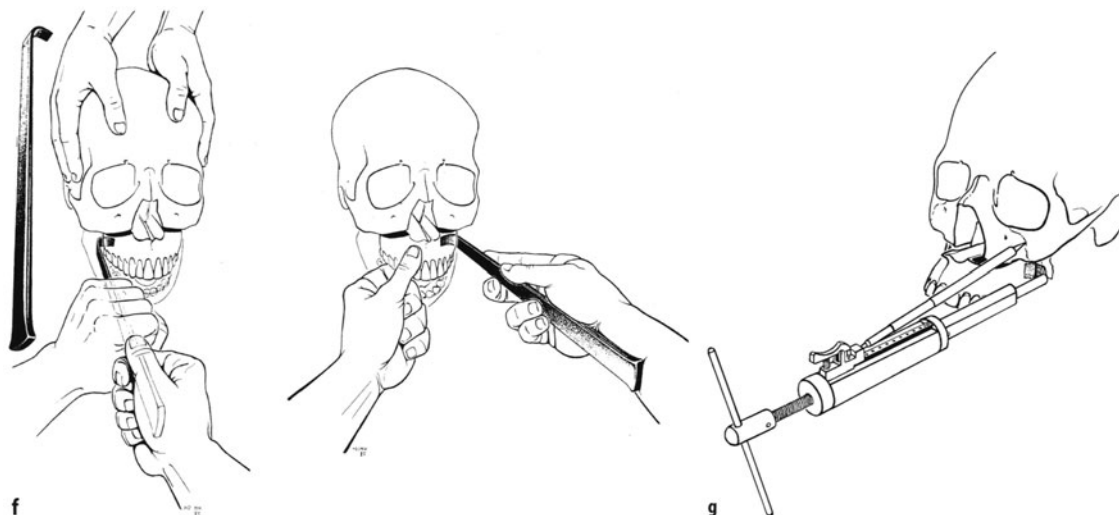


Fig. 96f,g. My final version of the Le Fort I-type mobilization. **f** Mobilization of the maxilla with the surgeon's hand power. **g** Mobilization of the maxilla with the maxillary advancer

part, a type of “shovel”, is inserted behind the tuberosity. That part can now be moved anteriorly millimetre by millimetre (Fig. 96g). After one side is done the next is dealt with in the same way. With the maxillary advancer one can move the maxilla anteriorly by more than 20 mm. One should not do as much as that on one side only, but go over to the other side after 10 mm or so of advancement. The maxilla must be so loose that it can be easily overcorrected with a pair of tweezers. It is a “must” (Fig. 96h).

Particularly in cleft cases, great resistance against the complete mobilization may be experienced. The palpating finger can identify where the knife or the scissors has to cut some still-resisting scars. It is better to cut them than to pull so hard with the maxillary advancer that they come loose. Scars do not include large vessels. So there is no risk of severe bleeding when cutting.

The maxillary advancer is also a very useful instrument for pulling the middle third of the facial skeleton forwards in a Le Fort III-advancement procedure.

21.6.1

Narrowing and Widening the Maxilla

Narrowing a too wide maxillary arch is not a difficult task: when the maxilla is loosened it can be approached from above. The necessary size of bone strip is removed with a bur from above, along or adjacent to the midline of the palate. The defect ends in the front between the two first incisors, or wherever planned.

Widening the maxilla is more delicate, at least depending on the amount necessary. For 2–3 mm only it can also be done from above (Fig. 96i). But if a widening of 3–5 mm on each side is required then the covering mucosa on the palate does not allow that much. In such a case I do it from below. I incise the palatal mucosal covering in the midline, diverging the incision from the papilla towards the lateral interdental spaces of the first incisors. The palatal mucosa is raised laterally, rather close to the sulcus of the palatine artery (Fig. 96j). The hard palate is cut from behind towards the interdental space of the two first incisors. A thin alveolar osteotome is used to split the alveolus in the front region while a flat twisting osteotome opens the two palatal defects. They should be filled with bone before the palatal flaps are re-sutured or held against the hard palate by a vaseline gauze pack. That gauze is held in place by a second plain gauze pad that has been moistened with acetone cement and fixed to the teeth with circumdental sutures that may extend across the palatal area on top of the gauze.

In such a case the maxilla must be freed all the way around, including from the pterygoid processes, but the nasal septum must not be detached and the maxilla therefore not mobilized, only widened. If the usual osteotomy along the hard palate were to be performed, that middle piece of palate would become necrotic because of lack of blood supply, as the nasal floor mucosa has to be raised for that osteotomy.

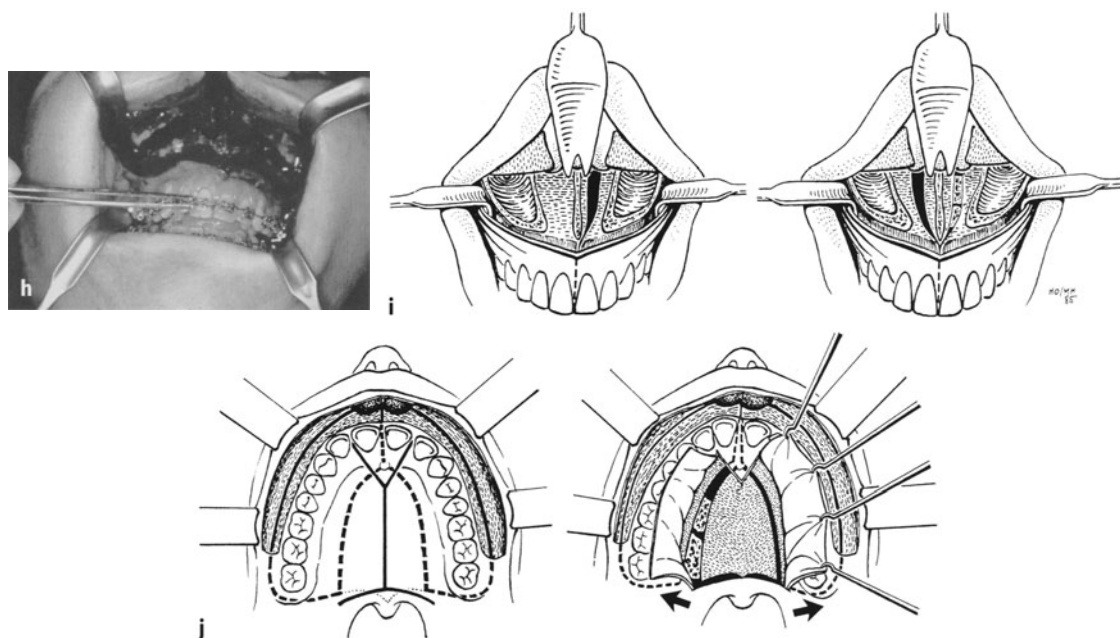


Fig. 96h–j. My final version of the Le Fort I-type mobilization. **h** The maxilla must be that loose that it can be overcorrected with a pair of tweezers. **i** Widening the maxillary arch from above when the maxilla is mobilized. **j** Widening the maxilla from below when a large amount of widening is necessary

21.6.2 Mobilizing and Repositioning the Maxilla in Sections (Fig. 97)

In some cases, in particular those in which presurgical orthodontics is not available, the maxilla must be mobilized and repositioned in sections in order to achieve the desired occlusal and aesthetic result. This is especially the case when the number of teeth present cannot be arranged into proper angulation to the respective base of the jaw without extractions. Mostly the front teeth are protruding and therefore require repositioning independent of the lateral parts of the maxilla. In these cases, a triangular osteotomy in the area of the necessary tooth extractions has to be performed with removal of a connecting strip of bone from the hard palate. That requires mobilization of the palatal mucosa from the front backwards, including the area of the connecting strip of bone which has to be excised. That means that for that frontal segment of the maxilla no palatal blood supply would remain. For this reason we must secure the vestibular blood supply to that anterior segment, exactly as in the operation procedure for correction of a maxillary protrusion situation only (S. Wunderer 1962).

The procedure is carried out as follows (Fig. 97a–e): the vestibular muco-periosteum has to be raised in the area of the horizontal osteotomy from the pyriform aperture back to the region where the wedge-shaped bone excision in the alveolus has to be performed. This undermining starts from an incision in the labial frenu-

lum and ends at a vertical incision in the vestibular mucosa where the bone excision in the alveolus, with or without extraction of a tooth, will be placed. From that medial incision in the vestibular mucosa the anterior nasal spine will be fractured or removed for better access to the nasal crest of the palatine bones and for easier elevation of the mucosa of the nasal floor. That anterior segment of the maxilla becomes free to be placed as planned, when the horizontal osteotomy at the anterior surface of the maxilla is done, the wedge-shaped bone is excised from the alveolus and the connecting bone strip on the palate is removed as well, after separating the attached portion of the nasal septum from the palate. If protruded teeth have to come into proper angulation, the freely movable anterior segment of the maxilla has to be raised in its posterior region. That requires gaining the necessary space by additional excision of the nasal septum or the nasal crest and of the posterior part of the horizontal osteotomy in the canine fossa.

In cases with a gothic-type dental arch, the mobile frontal segment has to be additionally split. This is performed from the palatal side where the raised palatal mucosa gives good access for doing this.

In addition to the freely movable anterior segment of the maxilla, the remaining posterior part of the maxilla may also need some widening or narrowing. This is achieved after that part of the maxilla has also been made completely free. If the necessary correction in transverse width is only minimal, it is achieved by cutting the remaining hard palate from above after it is

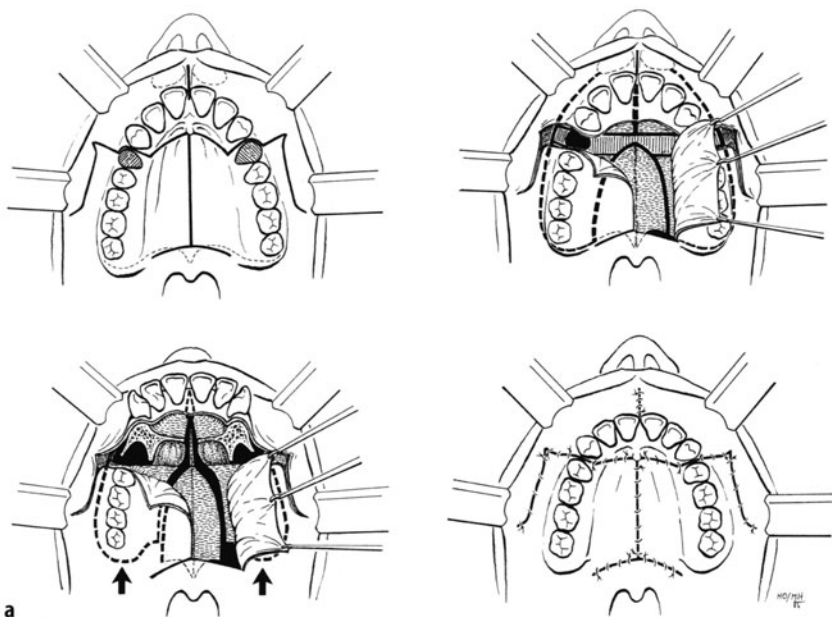


Fig. 97a. Mobilizing and repositioning the maxilla in sections for correction of dish-face deformity due to micro- and retromaxillism with anterior protrusion and lack of width of the maxilla. **a** Schematic illustration of the procedure for correcting the anomaly of the maxilla

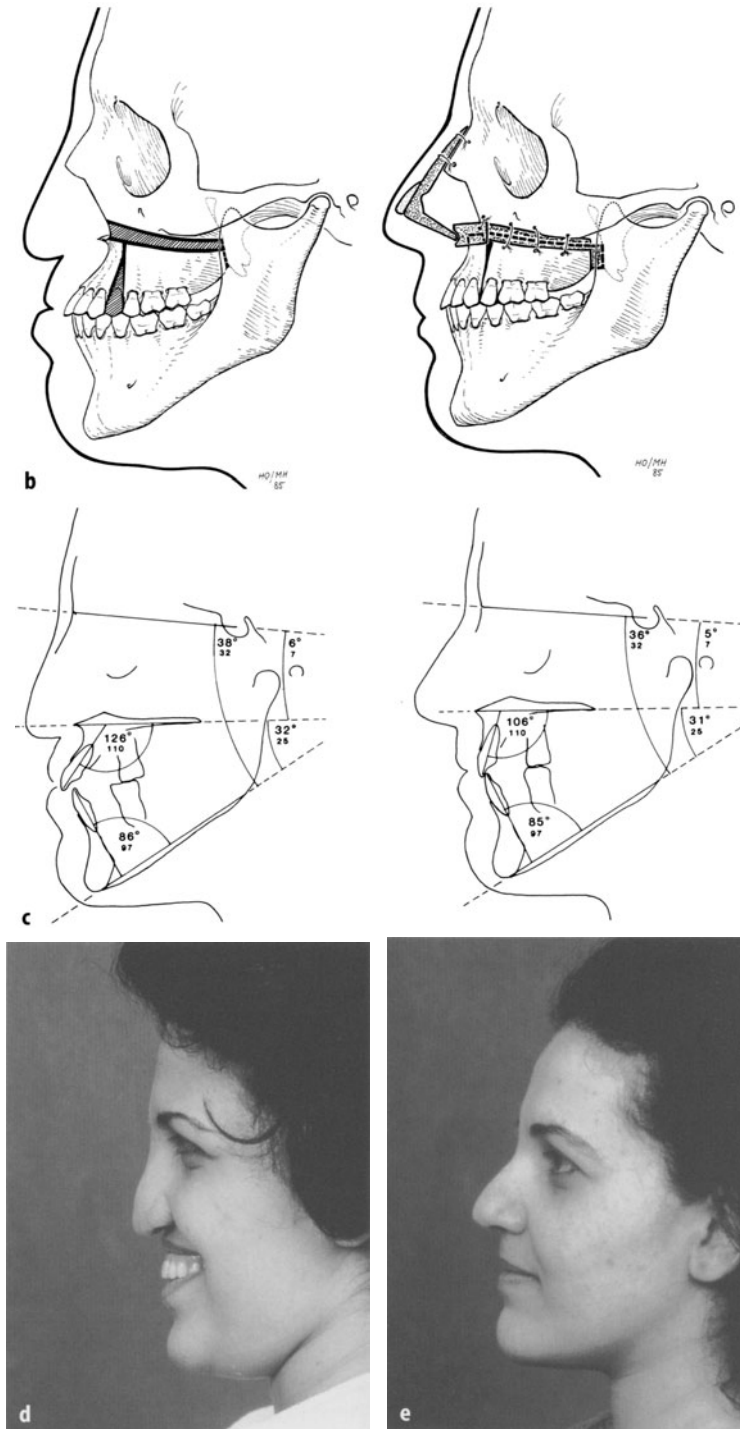


Fig. 97b–e. Mobilizing and repositioning the maxilla in sections for correction of dish-face deformity due to micro- and retromaxillism with anterior protrusion and lack of width of the maxilla. **b** Schematic illustration of the planned operation including reconstruction of the missing cartilaginous support of the nose by the use of an L-shaped piece of rib cartilage. **c** Tracings of the lateral pre- and post-operative cephalograms demonstrating clearly the result of the correction of the protruded upper front teeth and the retrodisplaced maxilla. **d, e.** Mobilizing and repositioning the maxilla in sections for correction of dish-face deformity due to micro- and retromaxillism with anterior protrusion and lack of width of the maxilla. **d, e** Patient's profile view before and 1 1/2 years after the correction

completely mobile. If a substantial widening is required the procedure shown in Fig. 96j will have to be applied, leaving the central part of the hard palate of that posterior half of the maxilla connected to the nasal septum and its mucosal covering. All four maxillary fragments are fixed together by a wire splint and, if prepared, with an additional occlusal splint or by miniplates and screws.

Mobilizing and repositioning the maxilla in sections can help to overcome the existing difficulties in producing a nice occlusal and aesthetic result.

21.6.3

Advantages for the Nose

The completely mobile maxilla offers the possibility of investigating the nasal cavity carefully. It is now easy to remove the often almost rectangularly bent nasal crest of the palatine bones. Particularly in cleft cases, it is often impairing the free airway in the inferior nasal choanae. Its complete removal during an ordinary approach for a nasal correction is often very difficult, rarely without causing severe damage to its covering nasal mucosa. It is much easier to free the periosteum including the mucosa from that irregularly formed piece of bone using the advantage of the access to the floor of the nose during a Le Fort I-type operation. This bone is exposed when freeing the nasal floor from its muco-periosteal covering and before that crest is cut with the forked septal osteotome (Fig. 98a).

Now, when the maxilla is mobile, there is the chance to see all details in that nasal cavity, with a good head light. Of particular importance is its floor, as bone fragments or severely damaged mucosa etc., need to be removed.

This access also permits very easy correction of a commonly occurring septal deviation. The septal cartilage is freed without any difficulty from its covering perichondrium including the mucosa (Fig. 98b). Any type of correction of a septal deviation is now done without difficulty. I correct its deviation by the use of the septal cutter (see Fig. 102e). Direct vision helps a lot in doing this.

The inferior turbinates also now deserve special attention. They can easily be reduced in size when necessary. In cases of correction of a maxillary long face, when the maxilla has to be positioned higher, these turbinates may block the inferior nasal airway almost completely when the mobilized maxilla is fixed in a higher position than it was before. The reduction in the turbinate's size is simple: a triangular piece of its inferior rim is cut out all the way back, preferably using diathermy. The defect should be closed with a resorbable suturing material. It is not advisable to reduce the turbinate's size with cutting diathermy without suturing. In the event of damage to the nasal mucosa, such a turbinate may become adherent to the floor of the nose and block the airway. Occasionally, although rarely, the turbinate including its bony part is resected.

Fixation of the Mobilized Maxilla

Next comes the fixation of the maxilla in the planned occlusion, followed by its osteosynthesis. Nowadays, plates are used by most surgeons. I am fond of screws or plates for the split mandible but not for the maxilla. By experience, I have learned that in almost 100% of the cases we will have a perfect occlusion after removal of

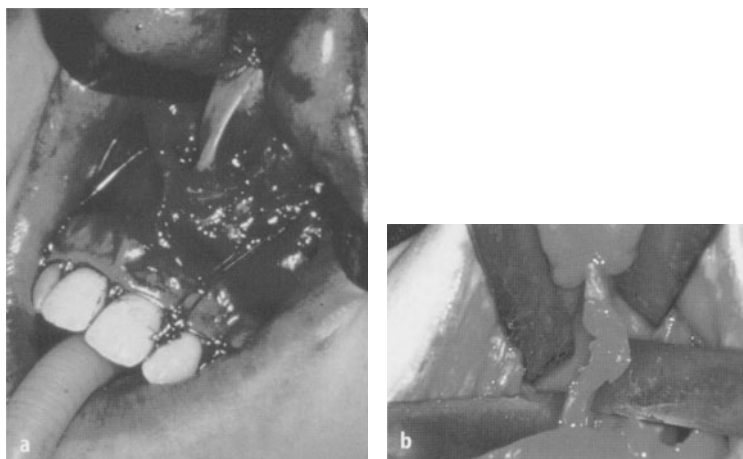


Fig. 98a, b. Correction of nasal septum pathology. The possible necessity for the correction of a septal cartilage deviation **a** and removal of the nasal crest of the palatine bones **b** should be evaluated in every Le Fort I-type mobilization. It is an easy task once the maxilla is mobile

the intermaxillary fixation as long as the patient was under general anaesthesia. But when fully awake and closing the mouth according to the muscular function, almost 50% showed a slight change in occlusion. This is also true for the mandible. With plates for osteosynthesis that is difficult to correct. Some surgeons leave them loose purposely. I prefer a pair of 0.4 mm wires on each side, one at the piriform aperture and one at the zygomatic crest, not tied too tightly. It will also produce bony union within 4–6 weeks, better without than with, intermaxillary fixation. The latter can prevent it and result in a pseudoarthrosis of the maxilla. But when necessary these wires permit postoperative correction of the occlusion by the insertion of elastics fixed to brackets on the teeth or the wire splints. For the mandible, there is no chance for such a postoperative correction of the occlusion if plates or screws have been used to fix the split mandible together.

Bone Grafting to the Osteotomy Lines

In cases in which bone grafting is needed in the canine fossa areas I will use three wires on each side which will prevent the bone graft from dropping into the maxillary sinus and from moving about.

For many years I used deep-frozen cancellous bank bone obtained from fresh cadavers. That worked nicely as long as the step or gap in the canine fossa was not more than 10 mm. Otherwise, the iliac crest is used to obtain the amount of cancellous bone required. For obtaining and preparing deep-frozen bank bone see Chap. 26.4.

The indications for using bone grafting in a case of Le Fort I-type mobilization vary, depending on whether or not enough bone contacts between the mobile maxilla and the upper osteotomy line exist without a bone graft and also on the size of the horizontal step or gap in the canine fossa. That defect may finally be bridged by scar tissue or a thin layer of spontaneously regenerated bone. In any case it will result in a flatness in that area, more so than it did preoperatively, when that gap was more than 8–10 mm.

Taking a bone graft is, of course, a disadvantage for both the patient and the surgeon. But the bone graft has the great advantage that it produces a nice contour, contrary to the otherwise flat-remaining paranasal-canine fossa region.

I also see the need for bone grafting when we have to produce a vertical gap in the canine fossa for the correction of a vertical maxillary deficiency (see Fig. 92e). In all cases of bone grafting to the canine fossa area, I also wedge a piece of bone into the empty space between the advanced tuberosity and the pterygoid process. There it does not need any fixation, but it fills the empty space, stabilizing the maxilla additionally.

21.6.4

Simultaneous Nasal Correction

The individual case may not only require maxillary repositioning but also nasal correction. It is more logical that minor or major nasal corrections be carried out during the same operating session, although never without the patient's presurgical agreement.

Once the maxilla is fixed in its new position then there will never again be an easier approach to the nose. The nasal crest of the palatine bones was removed while the maxilla was still mobile. Now the surgeon's favourite approach for nose correction is used and executed with the advantage that now the lateral nasal wall osteotomies can be performed under direct vision, using a rotating bur or a saw or even a nasal osteotome. The access to the nasal bridge and tip is the same as usually used by the surgeon. For smoothing uneven spots on the bridge I am very fond of the perforated nasal rasps as they cannot clog (see Fig. 102f). If there was also a septal deviation, that will have to be dealt with after the bony bridge and the work on the nasal tip is finished and before the vestibular incision is closed.

Finishing the Operation

The wound closure in the vestibulum of the maxilla is done with partially everted continuous suturing. I prefer a monofilament non-resorbable thread as I do not want the suture material to carry the mouth flora into the operation field. My favourite suture material for use within the mouth is Supramid.

The last step in the operation is to put a folded, rather small, soft rubber tube into the inferior nasal airways. This has the purpose of adapting the mobilized mucosa to the floor and lateral wall of the nose and also presses it gently against the nasal septum. In the case where a correction of a septal deviation has also to be performed, these soft tubes, in both sides of the nose, will guarantee that the cut septal cartilage will not swing to one or other side or become deviated again. I may change them after a day or two in order to get rid of the blood clots and then I leave them in place generally for 8–10 days in cases in which a septal deviation was corrected. It is essential to prevent these tubes from being aspirated. For that reason I fix them together with a mattress stitch to the columella. By doing so they can be left so short that they will not be visible. When no septal deviation had to be corrected I leave them or a strip of vaseline-impregnated gauze in place for 3 days only. It is obvious that the patient should not blow his nose for at least 12 days because of the open maxillary sinuses.

After every Le Fort I-type mobilization there is always a little bit of bleeding from the nose, both post-na-

sally and externally. After a few days it will look like bloodstained water.

21.7 Principal Complications, How to Deal with Them and How to Avoid Them

Problems During Surgery

Bleeding. It is the most common problem which can arise from behind the maxilla when the descending palatine artery in the retromaxillary area is cut with the osteotome when separating the maxilla from the pterygoid process. It is very rare in my experience. If it does happen it has to be stopped immediately by packing that area with gauze. After advancing the maxilla the vessel may become approachable for clipping. Otherwise a piece of bone is wedged in, or a vaseline gauze tampon is used. The end of the gauze strip should be left emerging through the sutured incision and gently removed within 3 weeks.

Ischaemia of the Vestibular Mucosa. It is not uncommon, in particular after the mobilization of the maxilla. In cleft cases, even the palatal mucosa can become blanched. This ischaemia can have two possible origins, either due to a local shock-like contraction of the vessels, partially also as a consequence of previous injection of a vasoconstrictor, or due to existing scars or damage to the palatal blood supply during the mobilization manoeuvre. For the latter reason the use of the maxillary disimpaction forceps was abandoned at my clinic after some bad experiences. When the ischaemia is due to previously existing scars on the palate or due to mechanical damage to the tissues, forget it and pray. I do not think anything will help, not even applying warm saline compressions with some Liquephine (antecoagulation fluid). When the ischaemia is due to a shock-contraction of the small vessels, application of warm wet gauze compressions will help, sometimes with some additional local injection of Liquephine at different sites. In these cases, the suturing of the wound edges has to be done as gently as possible.

Pulling the Maxilla or a Portion of it Right Out of the Mouth. It is the worst thing that can happen to the surgeon as well as to the patient! I have heard of such a misfortune several times, but never saw it happen. I can imagine that it could happen when pulling very hard to mobilize the maxilla with any type of instrument. For that reason one should not cut the maxilla into pieces before advancing it. When that is done it is more likely, that one-half suddenly becomes so loose that the surgeon cannot stop pulling. That should not occur when

mobilizing an intact maxilla. However, that too has happened. Avoidance of this is discussed under point 4.

Insufficient Mobilization. It is another problem. Many surgeons, in particular the inexperienced, hesitate to pull very hard. In cleft cases there is no other way to bring the maxilla forward than by using force. In order not to have a nasty surprise by its sudden loosening and then pulling it out of the mouth, all scars around the former operation area must be released by proper mobilization with a periosteal elevator or with a knife but not by elevating the palatal mucosal flaps. If the maxilla or its segments can only be brought into occlusion by force, then a relapse has to be expected.

To eliminate the risk of pulling the maxilla out of the mouth or insufficient mobilization, I had an instrument made by the Medicon Company, which enables the surgeon to pull the maxilla safely forwards, millimetre by millimetre. I called that instrument *the maxillary advancer*. It too requires that the osteotomies are completed all the way around the maxilla and floor of the nose. It also requires that all scarring in cleft cases is released. But, with that instrument, advancing the maxilla becomes a safe procedure. The maxilla must always be mobilized so well that it can easily be brought into occlusion or overcorrected, with a pair of tweezers.

Fracturing the Maxilla During Mobilization. It is another embarrassing problem. It easily happens when using two disimpaction forceps. A period of intermaxillary fixation will manage this, followed by the insertion of an acrylic splint.

Problems After Surgery

Quite a number are possible:

Late Bleeding from Behind the Tuberosity Area. I have seen this commence hours or days or even a full week after surgery. I have heard of cases in which the bleeding ended in the patient's death. One must reopen that region and pack it. Ligature of the external carotid artery may not stop the bleeding.

Infection. It can occur, usually caused by a retained piece of gauze. For that reason, in all orthognathic surgery only gauze with a radio-opaque thread should be used. Even an existing purulent infection of the maxillary sinus may not lead to infection when it contaminates the operating field during the Le Fort I-osteotomy procedure. It usually heals as there will be wide communication between the nose and the maxillary sinus. But gauze behind the tuberosity or in the antrum will always produce infection after some time. Besides gauze

left in the antrum, a piece of bone or bone graft in it gives rise to infection. Never have I experienced infection arising from a bone graft placed behind the maxilla. Whether or not the infection, often only a draining fistula, has its origin in a piece of gauze or bone, the actual cause has to be found and removed. A fistula in the former vestibular incision line can also occur due to a piece of suture material. That must be found and removed.

Partial Necrosis of the Vestibular Alveolar Mucosa. It is most likely to happen in cleft cases or when the blood supply is severely reduced due to shock or damage to the vessels. In such a case, the mucosa becomes pale, almost white. This was always a serious sign to me to stop the operation until the blood supply had recovered again. I applied wet, hot gauze, massaged the mucosa gently toward the incision line. Occasionally I injected Liquemine (an anticoagulant) into it, in several small deposits. Usually it started to bleed again. If finally the mucosa turned blue and did not recover, I went on with the operation and finished it. After surgery I applied vaseline gauze to the cyanosed areas and waited until secondary granulation out of the bone produced secondary epithelialization of the affected areas.

Partial or Total Necrosis of the Covering Soft Tissues of the Hard Palate. This is a very severe problem in cleft cases. I have had to deal with the consequences of almost complete loss of the palatal mucosa due to the use of disimpaction forceps. One of my trainees had tried too hard to get the maxilla loose in a cleft case, without freeing it properly from the surrounding scars. The use of disimpaction forceps was consequently abandoned in corrective surgery of the maxilla.

Partial Necrosis of the Maxilla or the Alveolar Segments. In a cleft case it is a very severe problem. It has its cause in an inadequate blood supply to the hard palate mucosa. Even in cases in which the palatal artery was ligated for primary closure of the palatal cleft according to G. Axhausen (1936a,b), there is still no risk in bringing the maxilla forwards. This is also true for those cases in which the descending palatine artery was damaged and ligated during surgery. R. Drommer (1979) has carried out a very valuable investigation on that subject. In 10 patients with cleft lip and palate he found, through angiography performed preoperatively, that the calibre of the palatine artery was considerably diminished. In several cases where the artery had been ligated we did advance the maxilla of these cleft cases in one or two pieces without any loss of mucosa, bone or teeth. This indicates that the blood supply from the soft palate is good enough to permit the palatal mucosa and the mobilized maxilla to survive. But this is only true when, during surgery, that palatal mucosa was not compressed or dam-

aged and when there are no other scars present other than the one in the midline. When the hard palate is very scarred due to several secondary closure operations on the cleft, it is very dangerous to perform the operation in the usual way with an almost circular incision in the vestibulum. For such a case, I seriously recommend that vestibular muco-periosteal flaps are tunneled but remain attached to the alveolar process and the horizontal osteotomy is performed through that tunnelling in the same way as I did when I started to develop the procedure (see Fig. 88a).

Obstruction of the Nasal Airway. It is another possible postoperative problem. It happens mainly when the maxilla is raised and the surgeon has not reduced the size of the inferior turbinates and did not correct an existing deviation of the nasal septum. This will have to be done as a second procedure. It can also happen when removal of a packing in the epipharynx was forgotten.

Obstruction of the Lacrimal Duct. It is another problem, rather annoying for the patient. It can happen only when either the osteotomy through the lateral nasal wall was performed above the inferior turbinate or when the duct was compressed by fragments of bone. The duct must be freed of its obstruction, if necessary by a dacryocystostomy.

Tooth Damage. It may also occur, sometimes like serial apicectomies. It is the surgeon's fault when he performs the osteotomy cut in the canine fossa too inferiorly. Endodontic treatment will become necessary.

Relapse. It has been a common problem for many surgeons. I was not surprised when I had watched others carrying out the maxillary advancement procedure. There are two very important principles to be observed in order to obtain stable results. The one I formulated: *"In orthognathic surgery the segments must be made so loose that they can be easily overcorrected with a pair of tweezers"* (Principle No. 50). If you need force to pull the maxilla into the planned position relapse will occur, in spite of the use of plates. The second principle in advancing and also in lowering the maxilla says: *"Gaps between bone segments should always be filled with bone"* (Principle No. 62). I had cases in which I advanced the maxilla by 2 cm and placed it 8 mm inferiorly in the front area and had no relapse. But I do believe in these two above-mentioned principles. It has been claimed that the lowered maxilla will not remain in its new position. I did not experience this when I wedged a piece of bone into the osteotomy line (see case Fig. 92). I also did not experience significant relapse when I repositioned the maxilla higher up, contrary to the upwards repositioning of the lateral maxillary segments for closure of an open bite, as suggested by K. Schuchardt (1954). The

difference in behaviour of the whole maxilla moved upwards and that of its lateral segments only seems explainable by the fact that in the first case, although we also reduce the size of the maxillary sinus, as is done in the second case, we leave a broad communication to the nasal cavity.

Flaring of the Nostrils. It often occurs after repositioning the maxilla cranially. This anatomical change may also be observed when the maxilla is advanced by more than a few millimetres and even more so with simultaneous widening in the front region. The cause of the flaring produced by these movements is related primarily to the change in surface area and position of the bone supporting the nasal alae while the upper parts of the nose remain in their original position.

To prevent or minimize nostril flaring, the soft tissues around the pyriform aperture must be completely mobilized. Recontouring the bony perimeter of the aperture also helps. It has been suggested that recontouring and reduction of the inferior rim after initial exposure has several advantages. By reducing the height in this area, the original vertical dimension of the aperture is maintained after superior repositioning of the mobilized segment(s). This also reduces or eliminates the ledge of the lower rim providing more direct access for atraumatic elevation of the muco-periosteum of the nasal floor, the lateral nasal walls, the nasal crest of the palatine bones and finally the nasal septum. This exposure also allows a more controlled separation of the nasal septum from the nasal crest of the palatine bones.

After maxillary mobilization the septum may have to be reduced vertically to prevent buckling with the upward movement. In addition, septal deviation, when present, is always corrected at the same time.

After the maxilla is fixed in the planned position, the lateral aperture rims are recontoured to eliminate any steps, restore symmetry and re-establish the original aperture size.

The mobilization of the soft tissues, bone reduction and recontouring, nasal septal vertical reduction and septal deviation correction, when present, all contribute to less distortion of the soft tissue and nasal flaring.

When flaring occurs in spite of the preventive measures described, medial rotation and fixation of the nasal alae can be accomplished by the use of fixation sutures anchored to the anterior nasal spine area. Prophylactic prevention of the flaring by the described procedures at the initial surgery is more effective than later interventions.

Bulky Cheeks in the Infraorbital Region. It can result when the maxilla is repositioned upwards by 5 mm or more. The best way to prevent this is by sufficient mobilization of the periosteum with the overlying soft tissues

up to the infraorbital rims and onto the bony nasal frame. When this has not been done and there is soft tissue bulk in the infraorbital regions, there may be spontaneous resolution with time which may extend over several years. I have observed this happening. However, when bulky cheeks persist and are a cosmetic concern for the patient, correction involves tissue mobilization as described. The prophylactic preventive procedure as discussed when accomplished at the original surgery provides a more predictable result than a later secondary correction.

21.7.1 Influence on Speech Quality

It is to be expected that advancement of the maxilla may have a significant influence on the patient's speech quality, in articulation as well as in the nasality of the speech. Particularly in cleft patients, the change in shape of the upper dental arch and its new relationship to the arch of the lower teeth in its new form will be strange for the tongue in its speech function. To a certain extent this will also be true for non-cleft cases. It is astonishing how much the articulation does improve when a retropositioned and collapsed upper jaw has been made into a normal dental arch and brought into normal relationship with the lower teeth. A formerly almost incomprehensible speech quality may become normal and easily comprehensible in spite of a still existing open nasality.

Advancing the maxilla may also influence the pharyngeal seal of the soft palate. In my own experience, I never found a permanent open nasality in a non-cleft case after advancement of the maxilla, not even when it was repositioned anteriorly by 15–20 mm. But I have experienced, although very rarely, a slight temporary open nasality in such cases approaching 20 mm, when the maxilla was advanced. In cleft cases the situation is different. There is always a median scar in the soft palate which does not permit it to adapt to the new situation as easily as in non-cleft cases. When no open nasality was present preoperatively in cleft cases, a slight naso-pharyngeal incompetence could occasionally be noticed, not only depending on the amount of advancement of the maxilla but also on the quality and the length of the soft palate. In these cases logopaedic therapy did help a lot. In those cleft cases in which some open nasality existed before the advancement of the maxilla, it happened rather often that the degree of open nasality increased, occasionally so much, that a pharyngoplasty became necessary.

The logopaedic team with whom we had the privilege to cooperate (C. Schwarz and E. Gruner 1976) carried out logopaedic examinations of over 100 patients from 1972 to 1974. From this clinical material, 40 cases were fully documented so that a comparison of the pre-

and post-operative speech quality could be established. Out of these 40 cases, 31 were cleft patients. These logopaedists came to the conclusion that “apart from the aesthetic, masticatory and psychological advantages

produced by maxillary advancement, the speech therapist can also recommend this operation from the logopaedic point of view, supported by the results of pre- and postoperative logopaedic investigations”.

The Transoral Chin Correction

22.1

Historical Background

When I started my training in maxillofacial surgery, in very severe cases only did surgical correction of the chin anomaly seem to be indicated. In the retrogenia cases, a bone graft was onlayed via a submental approach. It often produced, temporarily, quite a pleasing enlargement of the chin prominence but at the expense of possible infection and a submental scar which could be rather unsightly. And, of course, also there was the disadvantage of a second operation at the donor site for the bone graft. But there was another great disadvantage. It was the amount of resorption of the bone graft which followed within a year. And it was not easy to shape it so that it would fit nicely against the chin surface and it was not easy to fix it either. Often enough the bone graft was just placed onto the cortical surface of the chin, with some cancellous bone placed around the contact area. The experience with these onlay grafts led me to formulate the principle: *“Cortical bone shrinks like a mushroom in the sun, and cancellous bone melts away like ice cream when used as an onlay graft for contour correction”* (Principle Nr. 18).

Besides autogenous bone, autogenous rib cartilage was used and also preserved bovine cartilage. The first again had the disadvantage of the need for a second operation site on the patient and the second quietly disappeared.

Several types of alloplastic materials were used, such as titanium mesh (K.H. Thoma 1948) or acrylic. At the Maxillofacial Unit of Graz University, we had a number of different sizes and shapes in stock. We also tried to form them preoperatively in wax according to the patient's requirements and have them processed in acrylic, and implanted them also from a submental approach. It was not really successful. Too high an incidence of infection, secondary displacement, dehiscence of the suture line etc. did not allow it to become a practical procedure. The prefabricated silastic chin implants did much better but also often with too high an incidence of problems. It could even erode into the bone and come to rest against the roots of the patient's front teeth due to the pressure from the advanced soft tissues. J.M. Converse (1964) used the lower border of the re-

truded much-too-high (deep) chin as a free onlay graft, via an oral approach.

When I came to deal with such a case of hyper- and/or hypopretrogenia, I saw on the cephalogram that I could slide the lower border of the chin forwards and upwards, leaving it pedicled on the digastric and geniohyoid muscles. As I then did all mandibular symphysis and body fractures via the oral route, I also did chin advancement transorally, using J.M. Converse's degloving technique.

I cut off the lower border of the chin with a Lindemann bur. I was accustomed to using that easily breakable instrument from experience with it in other osteotomies. I directed the osteotomy line from low posteriorly to higher anteriorly. I could easily pull it forwards by 10 mm, pedicled on the geniohyoid muscles. In that first case I fixed the advanced chin horseshoe with a strong perimandibular Supramid thread on each side over an acrylic dental splint, so as to permit removal of the thread after three weeks (H. Obwegeser 1957) (Fig. 99a). The operation went without any problems and no postoperative complications. The result was very pleasing (Figs. 99b–e).

I reported my enthusiasm for this transoral chin correction procedure to my teacher Trauner and asked him by letter whether he knew of any literature on this technique. He replied no and wrote “but it is so simple that it seems impossible that nobody else had had that idea before”. Later, I used direct bone wiring for fixing the advanced horseshoe in the new position.

Soon, I learned that in some cases I had to make a more Roman arch out of a Gothic-shaped lower border or vice versa. And I also learned that, depending on the direction and shape of my bone cut, I could increase or decrease the chin height and could also correct a chin asymmetry (Fig. 99f).

In order to publish this valuable new technique more extensively (H. Obwegeser 1958) I tried to find anything similar in the literature. I found that O. Hofer (1942) had suggested sliding the inferior border of the chin forwards, from an extraoral approach leaving it muscle pedicled on the platysma, digastric and the geniohyoid muscles. His operation was performed on a cadaver using a rather large bone saw (Fig. 99g).

Later, my friend O. Neuner (1965) suggested doing a double-step advancement. I liked that idea and obtained

good results with it. In an appropriate case one can even perform a triple-step advancement (H. Obwegeser 1974). In order to handle the chin piece more easily, I started to free it from all musculature, trimmed and shaped it according to the requirements and then fixed it with direct wires. In a follow up investigation it was found that, on average, 50% resorption occurred, however, some of it was transformed into soft tissue, decreasing the amount of loss of contour in front of the bony chin. After that, we always left the advanced inferior chin border muscle-pedicated again, even in the double-step cases. A follow up study of these cases proved that there was only, on average, 10% resorption and even that amount was often found to be transformed into soft tissue, thus producing the planned amount of prominence advancement.

In double or even triple-step advancement, I often used deep frozen cancellous cadaver bank bone to fill the steps between the outer wall of the alveolar process and the advanced chin prominence. But even without it, I often found new bone formation in that step area.

22.2 My Final Method

There has not been much change since my publication in 1957 and 1958, except that the surgery has become less radical as far as bone exposure is concerned. If the chin advancement only is all the patient wants, it can be done as an outpatient procedure, under bilateral block and local anaesthesia. But there is no difference in the

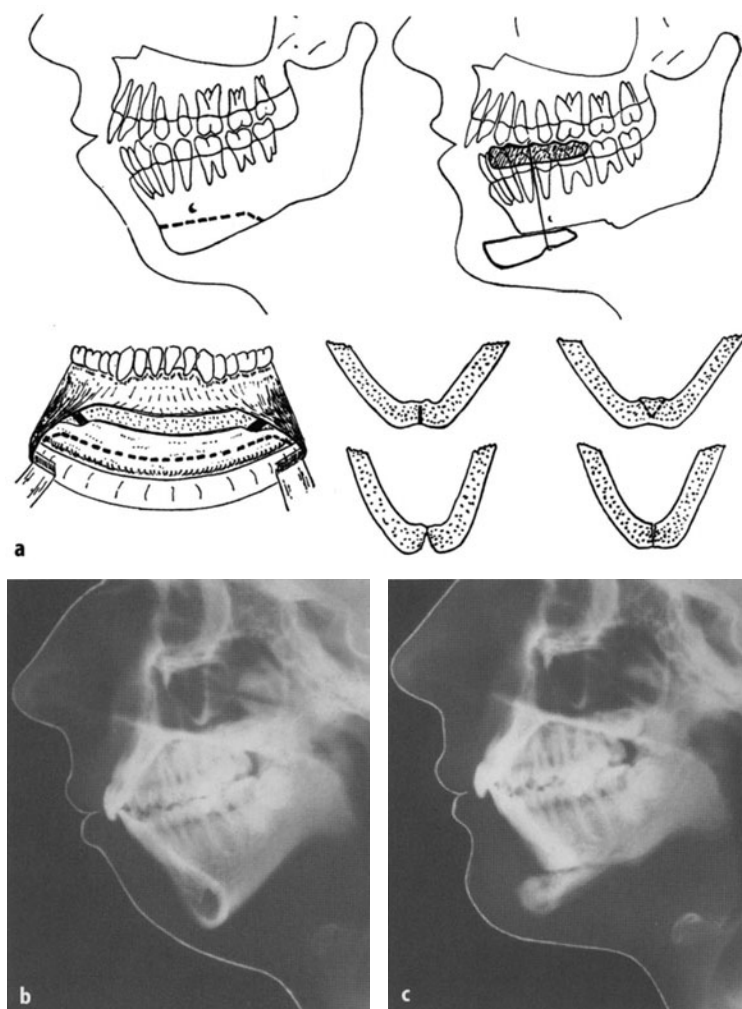


Fig. 99a–c. Transoral chin correction. **a** Diagrammatic illustration of first case of transoral sliding genioplasty (from H. Obwegeser 1957). **b, c** Radiographs of first case of transoral chin advancement (from H. Obwegeser 1957, 1958)

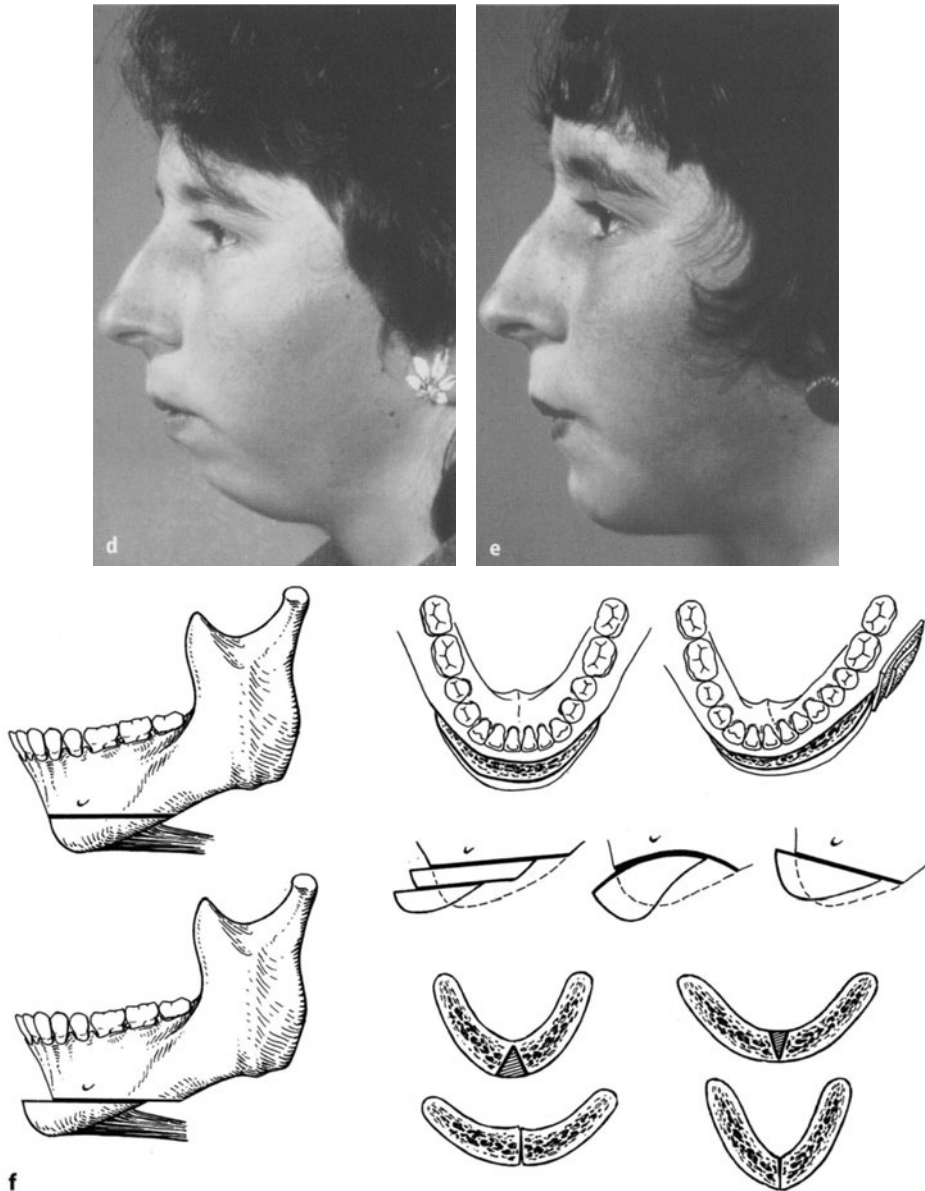


Fig. 99d–f. Transoral chin correction. **d, e** Profile views of first case of chin advancement, before and 1 year after surgery. **f** Schematic illustration of the various directions of the cut for achieving a sliding genioplasty to reduce or increase the vertical height of the chin prominence or to correct chin asymmetry and the radius of the chin prominence (from H. Obwegeser 1957, 1958, 1971)

operating technique whether the chin correction is performed under general or local anaesthesia.

The vestibular incision is made approximately 5–8 mm labial to the depth of the vestibulum, at a right angle to the mucosal surface only, and then directed horizontally to the alveolar process. The periosteum is incised from underneath the mental foramen as far back as necessary, from one side to the other. The direction of

the bone cut has to be determined preoperatively on a tracing of the lateral cephalogram, on which the desired profile line has been drawn. According to that planning, the lower border of the chin has to be moved forwards for correction of a retrogenia or upwards, if necessary even by excision of a strip of bone below the teeth, for correction of a severe hypergenia, or in a cranially convex curvature for the correction of a hypo- and retroge-

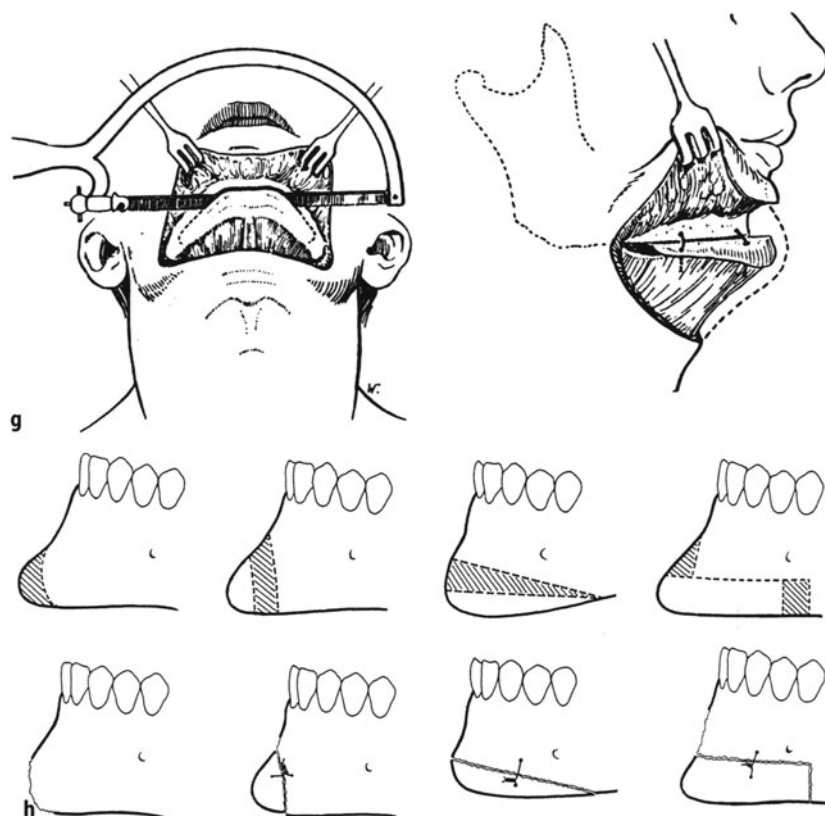


Fig. 99g, h. Transoral chin correction. **g** O. Hofer's suggestion for chin advancement, performed on a cadaver (from O. Hofer 1942). **h** Diagrammatic illustrations of various possibilities for reducing the size of the chin

nia. The existing shape of the chin and the result desired dictate the osteotomy cut. The tracing with the desired profile line permits the use of a transparent foil which is cut according to the existing shape of the bony chin to simulate the osteotomy line necessary to achieve the planned result. If more than 8 mm advancement is necessary, a double-step advancement should be planned. It gives a better result when each step is not too large. If there is some bone available to place on the step it is definitely an advantage. If there is a rather large step, the soft tissue line will show it in the final result as the soft tissue of the chin may be fixed into that step. The more the chin has to be advanced, the further back will I go with my bone cut, in particular when performing a double- or triple-step advancement. The mental nerve must be well protected. However, the complete degloving procedure belongs to history. The more soft tissues are left attached to the piece of bone which is to be moved, the less resorption of the moved segment will take place.

I personally like to use a reciprocating saw with thin disposable blades. My former co-worker, A. Triaca, does all osteotomies, even those for chin correction, with a

rather short, hard steel bur normally used in dental laboratory work (Maillefer Nr. 540). He does not free the prominence of the chin from the investing soft tissues completely. He leaves the inferior part of the mentalis muscle attached to it. He only frees the lower border in the area where the osteotomy reaches it. He resects the lingual rim of the detached chin prominence with the attached musculature using the same bur in order to reduce its backwards pull when the chin is moved anteriorly. This seems much better than dissecting the insertion of the musculature.

The technique for enlarging or reducing the width of the horseshoe-shaped lower border remains the same as published in 1958. However, it is not all that easy to excise a triangular piece of it at its posterior surface including the mental spine, and yet leaving some geniohyoid musculature in that area adherent. For that, the just mentioned resection of the lingual cortical rim helps to solve the problem.

When there is need to correct a hypogenia, again the desired profile line tracing is used to ascertain whether an upwardly-curved, almost semicircular bone cut will achieve the planned result or whether the sandwich

technique (J.M. Converse 1964) has to be used to achieve the necessary height increase.

The asymmetric chin prominence, not only present in cases of hemifacial microsomia but also in condylar hyperactivity cases, deserves special consideration. H. Sailer (1985) has suggested his so-called chin propeller technique. Another simple way is to cut the detached lower border into two unequal segments, using the symphysis as the site of the cut. Then the longer part is shortened so that from medial to lateral both are equal in length. Both segments are fixed together and to the chin.

Chin reduction is less often necessary than the correction of lack of its vertical height or horizontal length. The type of correction of surplus of the bony chin depends on its existing and the desired shape (Fig. 99h). To correct a horizontal surplus by trimming it off with a bur seems the easiest way. In my hands that very rarely produced a pleasing result. Almost always the prominence became too rounded. A much more pleasing result is achieved by a rather vertical strip excision. If the chin is too high (deep) in its vertical dimension preferably a wedge shaped piece will have to be excised, as shown in the illustration (H. Köle 1970).

The fixation of the advanced chin nowadays varies from surgeon to surgeon. Lag screws are rapidly inserted and quite sufficient. However, they do not need to be so strong that a horse could pull on them! Screws of 1.5 mm diameter should suffice. Others prefer specially designed plates. And it can still be done with wires if necessary, as I did for over 30 years!

The soft tissue surplus in the chin region is more difficult to correct than are the bony abnormalities. A certain amount of contour reduction can be achieved by reducing the underlying bony chin. But there are cases which definitely need soft tissue excision, skin as well as subcutaneous tissues and musculature (Smith 1985).

The method of managing and *closing the incision line* is important. I have often been asked what I do to prevent suture dehiscence which I have sometimes seen in patients of my trainees. There are a few important points to be observed. It begins with the incision. The knife must cut the mucosa at a rightangle to the mucosal surface. That permits good adaptation of the wound edges, much better than when the knife cuts through the mucosa obliquely. Secondly, with the subsegment vertical cut to the chin area, quite a bit of musculature remains on the chin. When closing the wound, this permits a better bite with the needle than when mucosal edges only are approximated. Thirdly and most important, is the handling of the edges of the mucosa. It is better not to use toothed forceps to hold the edges. Fine single hooks do less harm. Next is the type of suture material to be used. No hydrophilic thread is any good. It absorbs saliva and conducts infection into and underneath the approximated edges.

For more than 45 years, I have preferred "Supramid", a suture material which is not as stiff as a monofilic thread as it consists of a great number of very fine filaments which are covered by a layer of non-hygroscopic material. The type of suturing is also important. Sometimes I even used submucosal catgut sutures. I gave it up. They are too difficult to insert. I prefer a continuous suture, changing between a vertical mattress type to an occasional ordinary continuous suture. Very gentle adaptation is important. Postoperatively, I compress the soft tissues towards the chin, particularly above the advanced part, with some slightly elastic strips.

Since I published this transoral chin correction in 1957 and 1958 it has become a widely used procedure, also in connection with elongation osteotomies of the mandible (see Figs. 13+14). J.M. Converse, who had never done a sagittal splitting of the rami until we met, asked me twice where and how I obtain the necessary quantity of skin cover. I told him that, if necessary, I mobilize the periosteum bilaterally with the investing soft tissues around the chin and all the way back beyond the angle of the jaw.

22.3

Principal Complications, How to Deal with Them and How to Avoid Them

22.3.1

Problems During Surgery

Bleeding: Bleeding from the incision of the mucosa is rarely a real problem. Occasionally only a small vessel may become troublesome. Electrocoagulation will stop it.

More troublesome bleeding may arise from a vessel within the musculature which is attached to the lingual side of the chin. The reciprocating saw can cut too deeply into these muscles. The bleeding vessel cannot be seen as long as the chin prominence is not detached. Thereafter there is good access to clip or just coagulate the vessel. Bleeding occurs more often in a double- or even a triple-step advancement of the lower border of the chin. It should not be a reason for detaching the musculature from the chin horseshoe.

The Mental Nerve. It can be damaged when mobilizing the periosteum or when performing the bone cut with a saw or a rotating bur. Pressure or pull on the nerve may cause temporary hypo- or anaesthesia which recovers within a few weeks or months. Damage to the nerve by a bone cutting instrument may be due to partial or complete cutting of the nerve. In the first instance sensibility may return after 6 to 24 months, in the second, nerve anastomosis before wound closure may help, but only very occasionally. When the nerve is cut or pulled out of the foramen complete anaesthesia of that side of the lower lip will result. Almost always, in some cases after

one or two years, in other cases even later, some kind of sensation is felt by the patient. Some will even forget their numbness of the lower lip and tell the examining doctor that everything is normal. And yet, when carefully checked, normal sensation cannot be proven.

In some cases, in particular when infection at the site of the nerve has occurred, intractable pain will result. It is a constant type of neuralgia like a neuritis, in spite of complete anaesthesia of the lip. It is called “anaesthesia dolorosa”. Nothing seems to be able to cure that pain, not even high doses of vitamin B12, nor injection of concentrated alcohol into the mental foramen and also not the ablation of the whole mandibular nerve from the lingula to the mental foramen. For that type of anaesthesia dolorosa I do not have any other explanation but that it exists on the basis of nerve vessels. Even injections of local anaesthetic cannot eliminate the pain completely. They reduce it only for a short while.

Wrong Direction of the Bone Cut. It is not uncommon. The cause for it is inexact planning. When it happens try to make the best of the situation!

Problems With Fixation. They can hardly be expected when the chin prominence has to be fixed as one single piece of bone into the planned place, independent of whether the surgeon uses wire fixation, as we did for thirty years, or lag screw or plate fixation. Fixation becomes more difficult when a two- or three-step repositioning is planned and even moreso when the detached horseshoe has to be separated into two pieces either for reasons of asymmetry or in order to make a Gothic arch out of a Roman arch or vice versa. However, with direct screw fixation and with plates and screws this should not cause any real problem.

Losing a Piece of a Bur or a Saw Blade. It happens occasionally, but not when non-breakable saw blades are used. One should try to find the foreign body, but without risking detachment of the genioglossus and geniohyoid muscles from the bony horseshoe. Rather leave it and inform the patient. It will rarely give rise to abscess formation. If it does it must be removed.

22.3.2

Problems After Surgery

Infection With Slight Pus Discharge. This is seen rather often. In such cases, we routinely rinse the operating field twice a day, through the incision line in between the sutures. After a week or so the pus production will stop. If not, one will have to think of a forgotten gauze in the operating field or sequestration of the repositioned bone segment. In both situations the cause must be removed and the operating field washed thoroughly with a strong antibiotic solution and if possible, after spray-

ing some antibiotic powder over the whole operating field, the wound is closed again.

Suture Dehiscence. It seems to be a rather common problem. Secondary closure will rarely be successful. Daily rinsing with antibiotic solution and covering the wound with a vaseline gauze strip which is covered and soaked on its surface with an acetone glue. Once, on my ward round, I found quite a number of suture dehiscence cases after both sagittal splitting of the rami as well as after chin correction only. I was astonished and asked the trainee in charge what he thought of this. His reply was short and clear: “Don’t you know, professor, that this is quite common and does not matter?”. It means that it almost always heals without great problems, however, with additional treatment and costs for the patient. I prefer to prevent it by proper suturing as mentioned in the section “Closing the Incision Line”.

Relapse. That means displacement of the repositioned horseshoe. It can happen within the first three weeks, rarely later. It is due to inadequate fixation. I have heard of it, but never experienced it myself. A radiograph will give the information as to what has happened. Proper refixation is the necessary treatment.

Resorption. It will always take place to a certain extent, but maybe without spoiling the final result, since in most cases of muscle pedicled chin correction the average resorption of 10% is replaced by fibrous tissue. In cases when the resected chin prominence is repositioned as a free bone graft and not muscle-pedicled, the average resorption will be about 50%, as mentioned before. A part of that will again be replaced by fibrous tissue.

Unaesthetic Chin-neck Contour. That can easily occur when it already existed preoperatively and the chin prominence was moved forwards, pedicled on the platysma muscle. In these cases, I want to reposition the horseshoe pedicled on the hyoid musculature only, independently of whether it is a one-, two- or three-step advancement. Then the chin-neck line will always be better than when the horseshoe is left pedicled on the platysma muscle also.

Unightly Upward Retraction of the Skin Behind the Advanced Prominence. That can occur when it already existed before surgery as an ugly skin fold and the chin prominence has been brought forwards quite a distance. Although the skin will be rather tight behind the chin, in spite of that, an empty space can be palpated where the upward retraction was before surgery. In these cases I fill that area up with thin slices of lyo-cartilage via the oral access before closing the vestibular incision and obtained excellent results. Any other material for contour correction may be just as good.

22.4

Deep Frozen Bank Bone

The type of material to be used to fill gaps in the bone is worth mentioning in detail, as the same need exists not only for the sandwich chin enlargement but also for the Le Fort I-advancement or maxillary vertical height increase, etc.. Several materials have been used to fill such gaps. Best of course is the patient's own bone. Again deep frozen cadaver bank bone was used for the rather rare case of sandwich chin enlargement and, as mentioned earlier for maxillary advancement, with very good results as long as the defect was not larger than 10 mm. At that time we did not bother about tissue compatibility between recipient patient and donor. We never experienced any problems with this. We removed as much cancellous bone from a donor cadaver as we

could, under sterile conditions, rinsed it with an antibiotic solution, put it into a sterilized jar with a cover and then placed it into the deep freezer at at least 20° centigrade below zero. In the same way we used surplus cancellous bone or ribs we had taken from patients during surgery. Whenever there was need, we took it out of the deep freezer and implanted it sometimes more than two years after it had been taken. Today it is too risky to use deep frozen cadaver cancellous bone because of the possibility of various viral infections. One can obtain and use it only when that cadaver has been fully checked and approved as an organ provider.

Whether lyophilized bone is of any use for these purposes or not, I do not know. I can imagine that autoclaved bone could do very well for such purposes, as long as the gap is not more than 10 mm. It might be worthwhile to have it tested on animals first.

Masseter Muscle Hypertrophy and Bony Surplus

Recently I have been asked by several quite experienced surgeons how I corrected masseter muscle hypertrophy. I was surprised to learn that an extraoral approach was still considered to be the best access for dealing with it. This has also recently been suggested in a textbook. For those reasons there is obviously an indication to have some discussion on this subject also.

23.1 Clinical Findings

This condition can be unilateral or bilateral. It may be almost symmetrical or with quite some difference between the two sides. The hypertrophy of the chewing musculature may be limited to the masseter muscle only or others as well, such as the temporalis, the pterygoids and even the sterno-cleido-mastoid muscles (T. Iizuka 1992).

In most cases, the patient seeks help for aesthetic reasons only, while in other rather severe cases patients complain of reduced mouth opening and even pain when chewing. The patients do not know why and when the condition started. It may lead to bruxism and TMJ problems and even to reduced mouth opening.

Because of the possible variations in signs, the investigation must also include palpation of the other chewing muscles and if necessary even clarification by using CT or MRI techniques. These diagnostic studies must include examination of the TM joints, clinically and in TMJ pain cases in even more depth. Often enough, masseter hypertrophy is not just a simple problem of surplus of the masseter muscle. Whether uni- or bilateral, the abnormality may be limited to the muscle only or also include a minor or major angle flaring.

It is not known why it occurs. Histologically the muscle fibres do not show any specific abnormality. Histochemical investigation revealed that in the majority of cases the larger type I fibres predominate while the type II fibres were fewer than normal. It is anticipated that the surplus of muscle may have its origin in the transformation of the smaller type II fibres into the larger type I fibres (M. Farmand et A. Railerson 1985).

23.2 Historical Background

According to the literature, there are, or at least have been, several ways of dealing with the surplus of the masseter muscle. Either, via an extraoral approach, a somewhat triangular inferior portion of the volume of the muscle was excised (D. Dencer 1961; W. Wade and E. Roy 1971); or almost half of the muscle, on its inner aspect was removed (W. Adams 1949; F. Masters et al. 1955); or the muscle on its lateral side was excised (F. Ginestet et al. 1959). A reduction in muscle volume and bone via an oral access was reported by J.M. Converse in 1964.

It was in 1953; I was already a few years into my specialization training, when a 22 year old female patient turned up in our outpatient clinic at the Maxillofacial Surgery Clinic at Graz – University Hospital, seeking information on what could be done for her bilateral cheek swelling. She had typical bilateral masseter hypertrophy. She came directly from the adjacent ENT-clinic which she had attended for the same reason. There, she was informed that the swelling was purely muscular and could be corrected. For the correction, a submandibular incision would be necessary to approach the mandibular angle area where the muscle was attached. The surplus of that muscle would then be excised. She was informed of some possible complications of that extraoral approach. As she did not like the idea of that submandibular approach she sought further information from another institution.

As I had already had some experience with approaching the mandible via the oral route through transoral open reduction of fractures of the mandible, including the angle regions, and my sagittal splitting technique on the ramus, I did not see any need for an extraoral approach. Instead, I suggested performing the reduction of the muscle surplus via the oral route with almost no possible complications. She liked the idea of having it done without the production of a submandibular scar. According to the anatomy it should not be too difficult a procedure to remove the muscle surplus between the periosteum and the lateral layers of the masseter muscle and also the bony surplus in the angle region, via the oral access. The patient agreed and she

and I were pleased with the result. I never did a case using an extraoral access. Some months later, I left for Zürich. I never found time to publish this, but I taught it to my trainees and the many guest visitors.

M. Perko (1963) had published on that subject after his stay at my former institution in Graz, Austria. He mentions that he does the reduction of the muscle surplus via an oral approach, while for the correction of the bony surplus, an additional extraoral incision is necessary. His teacher, F. Celesnik in Ljubljana, Slovenia, was, like me, also almost a pupil of Trauner, through his several visits to the Graz school of maxillofacial surgery. So, Perko and I had the same philosophy on many subjects in our speciality. For that reason I asked him to join me at the clinic in Zürich. He was a brilliant surgeon. His main interest was primary cleft work.

There was a short publication on the reduction of bilateral hypertrophy of the masseter muscles via the oral route by G. Hankey (1968). He did it with the help of G. Seward. Whether Seward had seen it when he stayed with me for a longer visit or whether it was his own independent idea I do not know.

H. Beckers (1977) described our procedure, step by step, and reported the results and the problems experienced with it, based on 17 cases at our clinic, when he trained with me. There has not been much change since, except the improvement in instrumentation. T. Iizuka (1992) has written a fine chapter on this subject with excellent results, having learned the procedure at my clinic when he trained with me for two and a half years.

23.3

My Technique Since 1953

The average case does not produce any difficulty as long as the anatomical structures in the vicinity of the masseter muscle are kept in mind. It is the more asymmetrical case with an abnormal quantity of bone surplus which requires a certain degree of experience and surgical skill and, in addition, good instrumentation.

The procedure is easily understood from a good diagram (Fig. 100a). For the removal of a rather well flaired angle, I personally prefer to use the reciprocating saw. That permits the surgeon to cut off the angle area horizontally. To do this with a bur is much more difficult. With especially long and very thin, flexible, non-breakable blades, the bony surplus in the angle area is easily cut off, whether it protrudes laterally or downwards. These blades had been specially made in different shapes for the purpose of working on the maxillofacial skeleton. They make it rather easy to cut off the whole lower border of the mandible, as is often necessary in H.H. cases (Fig. 100b).

The bilateral symmetrical cases are not that difficult for production of a nice symmetrical result (see Fig. 44). The very asymmetrical case of muscular as well as of

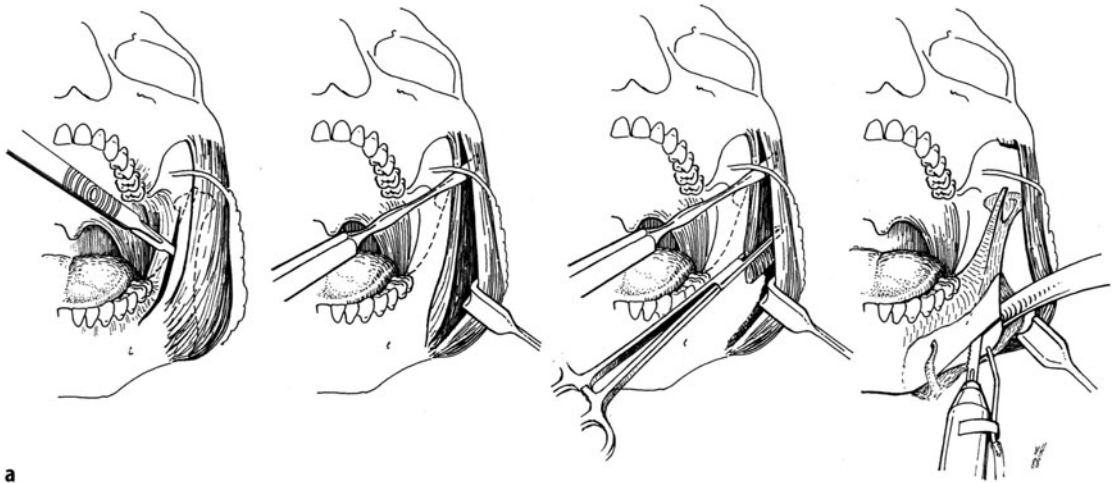
bony origin requires quite some experience to produce a good symmetrical result without any complications. In such cases it may not be enough to reduce the amount of muscle mass of the masseter that one feels is necessary and grind or cut off the protruding portion of the angle region. Such a case may, in addition, require a full decortication of the severely laterally projecting part of the mandibular body as is occasionally the case in unilateral H.E.-deformities (see case Fig. 54).

A rather unusual case is used to demonstrate the procedure. The 30-year-old male patient reported that his face had been asymmetrical since birth. He had never thought of having it corrected. But over the last half year the left side had become bigger. That was the reason why he now wanted to know what kind of a swelling he had in his two mandibular angle areas. He said that the swelling on the left side had recently increased and had become harder. He had no problems with mouth opening or with chewing. Apart from this anomaly he did not know of anything else wrong with his health.

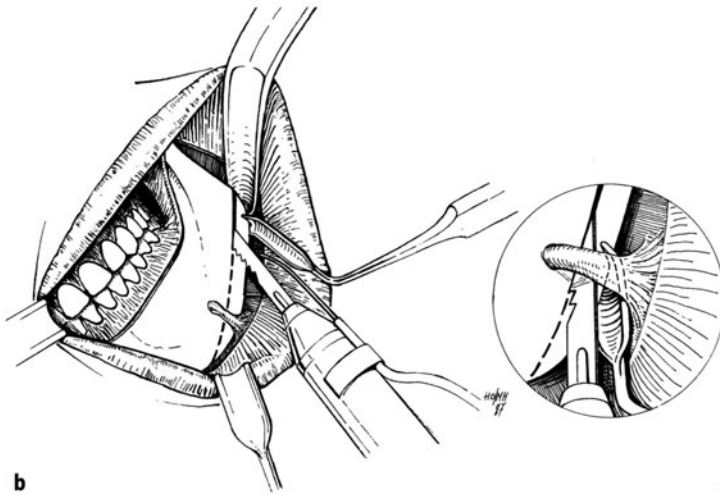
The clinical investigation revealed nothing else but the localized swellings of a rather hard consistency in the angle areas. When pressing the teeth together they increased a bit in size and hardness. They were of a rather unusual shape (Fig. 100c,d) and more visible from below than from in front.

Radiological examination revealed the typical picture of flared angles in a masseter hypertrophy condition, as normally seen in the panoramic view (Fig. 100e). The p.a.-projection of the mandible in the open mouth position revealed two entirely different angle – ascending ramus situations (Fig. 100f). The right was practically inconspicuous except for a small angle protuberance. The same parts of the mandible on the left side seemed broader in the whole ascending ramus – angle region. Whether this was due to actual bone volume or due to an outward rotation of that area cannot be determined from this radiograph. It definitely influenced the facial contour. The horizontal CT of the mandible (Fig. 100g) did not show any increase in bone volume, but a larger masseter muscle on the left than on the right side.

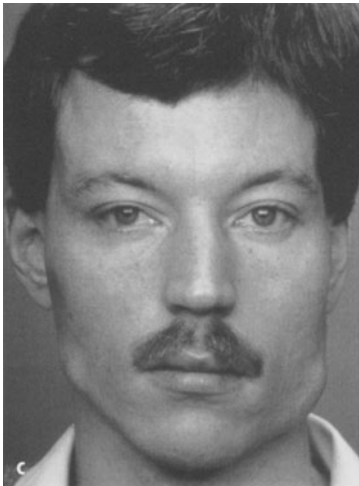
The procedure is done as follows: Before surgery starts, a cortisone derivate is injected or any other medication is given which will reduce postoperative swelling. This is repeated after surgery or 3–4 hours after the first medication. Under transnasal-tracheal intubation anaesthesia and with a medium-sized mouth gag on one side, either under hypotension or after injection of a vasoconstrictor, the ramus is approached similarly to the sagittal splitting procedure. For the resection of muscle surplus only, the incision may start in the first molar region. If bony surplus also has to be removed, I start at the incisor region as I wish to visualize the mental nerve. The incision extends to the base of the coronoid process, but does not include the periosteum.



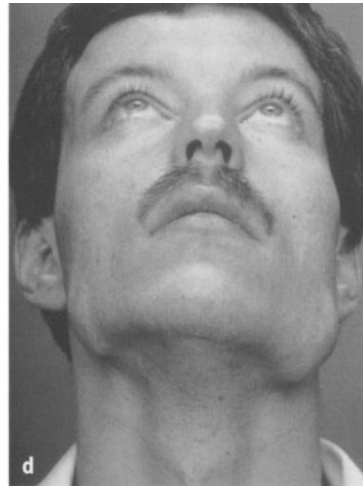
a



b



c



d

Fig. 100a–d. Correction of masseter hypertrophy. **a** Diagrammatic illustration of the steps of the procedure for muscle and angle reduction. **b** Cutting off the protruding angle or the whole inferior rim of the mandible using a reciprocating saw. **c, d** Atypical swelling in the mandibular angle regions in a 30-year-old male

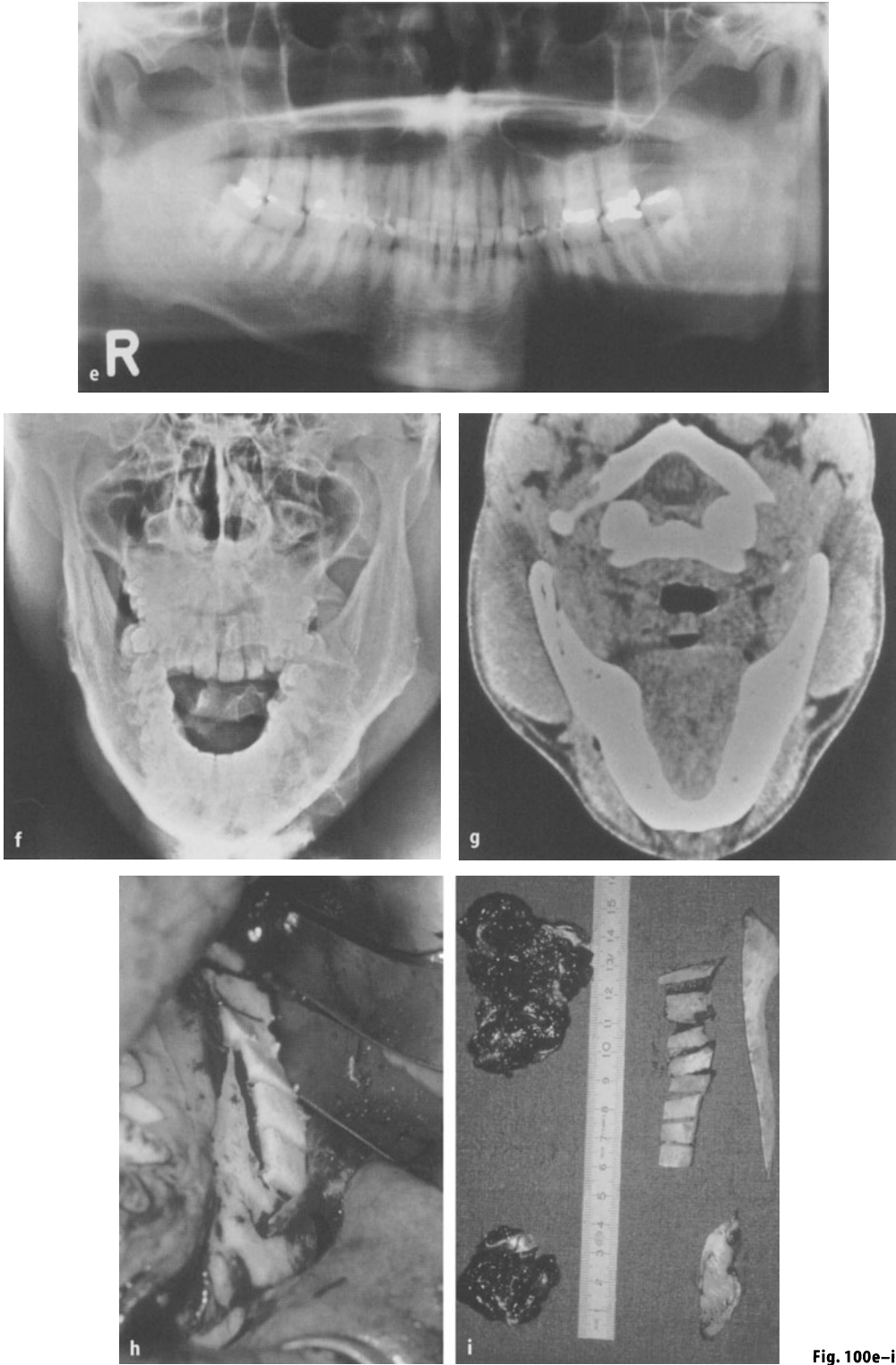


Fig. 100e-i

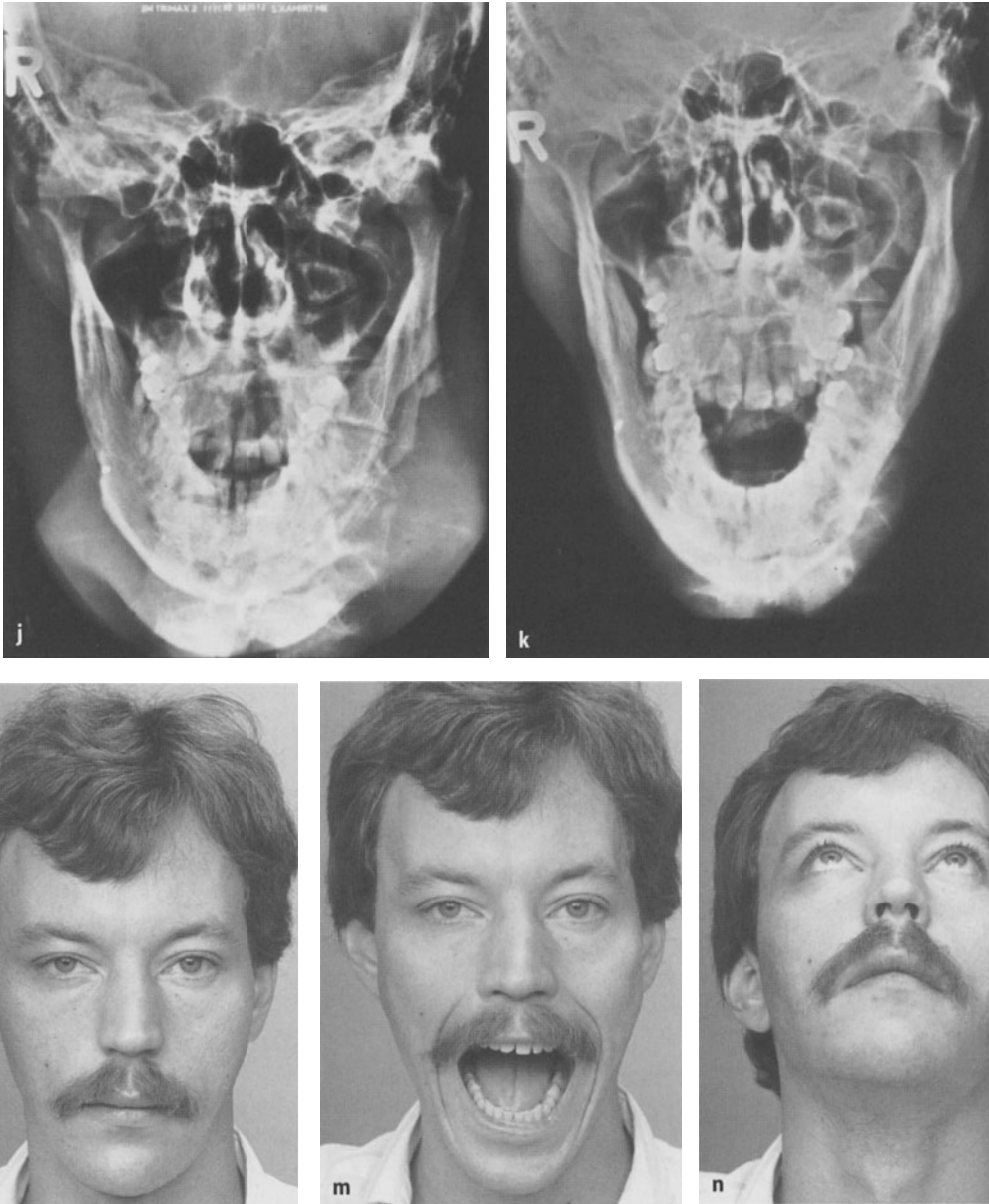


Fig. 100e–n. Correction of masseter hypertrophy. **e** Panoramic view showing typical shape of the angles in masseter hypertrophy. **f** Mandible p.a. projection in the open mouth position discloses a much wider left angle and ramus area than the right and as normally seen. **g** Horizontal C.T. reveals a larger right masseter muscle than the left, but nothing of the bony differences between the two sides. **h** Strip decortication to remove the lateral bony surplus. **i** Different quantity of muscle and bone after removal of the surplus on the two sides, depending on the presurgical situation. **j, k** In the p.a. projection of the open mandible the lateral mandibular aspect looks very irregular after the extensive decortication, but 5 months later it is almost equal to the other side. **l–n** Patient's appearance and normal mouth opening 5 months after surgery

The soft tissues are dissected from the periosteum along the lateral side of the anterior ramal crest until the knife reaches the masseter muscle. This dissection extends just to the semilunar notch level and down to the lower border in front of the attachment of the masseter muscle. Its fascia is incised, from the zygoma all the way down to the front of the angle where the muscle inserts. The fascia is raised laterally. Next, the muscle is separated from the periosteum on the lateral aspect of the ascending ramus with careful sharp dissection or by blunt dissection, extending upwards to the zygoma and the zygomatic arch and down to the insertion of the muscle above the angle of the jaw and backwards to the posterior border of the ramus, but no further. The dissection of the soft tissues is carried forwards and down to the lower border of the jaw in front of the muscle. The muscle must be visualized in its full length and thickness. It is not that easy to differentiate between the vertical and the oblique parts of the muscle. The soft tissues of the cheek are not separated from the muscle, as the parotid duct and some branches of the facial nerve could easily be damaged there.

The surgeon will now have to decide how much of the masseter muscle he wants to resect. That may vary between $\frac{1}{3}$ to $\frac{2}{3}$ of the whole thickness of the masseter muscle. A broad periosteal elevator or a pair of undermining scissors follow the muscle parallel to its outer surface, at the desired distance, up and down. The periosteal elevator marks the inferior level of the part of the muscle to be resected. With it in place, an artery clip with long handles grasps that muscle portion at its lower end. With a pair of curved scissors or a knife with a long handle this portion of the muscle is dissected from its insertion above the angle; while the periosteal elevator and the assistant's retractor ensures that the remaining lateral portion of the muscle is not cut. With the clip still holding the muscle portion to be resected, the periosteal elevator follows its lateral aspect up to the zygomatic arch. Another clip now grasps the dissected muscle portion at its upper insertion. Next the muscle is cut above the clip along its upper bony insertion, bit by bit, taking care that the cutting instrument does not cut anything behind the ramus and above the semilunar notch. There it is dangerous. Bleeding may occur, difficult to clip, and the facial nerve is not too far away from the posterior rim of the ascending ramus either. Because of the facial nerve, the assistant holding the remaining lateral part of the muscle out of the way, should not pull too hard on it laterally. Ordinary flat soft tissue retractors with the end bent downwards should not be used here. Much safer are the channel retractors with an upwardly bent end.

If bony surplus is to be removed also, then the next step in the procedure has to follow. This will be done in an almost closed mouth position. As mentioned before, the incision in the vestibular mucosa runs forward to

the incisors, of course, in the movable region. The periosteum with the soft tissues is elevated down to the lower border and posteriorly to the muscle insertion. Care must be taken not to damage the mental nerve. The periosteum is then raised gently around the lower border and around the angle and also half way up the posterior border of the ramus, leaving the pterygo-masseteric sling intact, in order to avoid the remaining part of the masseter muscle retracting upwards. This detaching of the muscle sling around the angle has to be done very carefully. It is best done by the additional use of the lower border periosteal elevators.

Depending on the amount and location of bone to be removed, it may be simply done with burs, chisels or a reciprocating saw. For that type of work, the upwardly bent retractors or the mandibular channel retractors are inserted to hold the periosteum and the remaining masseter muscle out of danger from the bur or the saw.

As mentioned earlier I prefer the specially designed long, non-breakable blades in the reciprocating saw since they are available. With these, one can either come from above the mental nerve to cut off the angle or even from underneath it, working parallel to the inferior rim with the saw blade vertical to the bone (see Fig. 100b). To reduce the surplus of the lateral aspect of the mandible, a decortication is performed. Whether one takes off the lateral cortical plate in one piece (less vertical cuts, less risk of damaging the mandibular nerve) or as a strip-by-strip decortication (Fig. 100h) depends entirely on the surgeon.

The excised muscle and bone quantity may differ very much from one side to the other (Fig. 100i), depending entirely on the presurgical situation.

Before closing the incision, the operating field is rinsed with an antibiotic solution and closed with a vacuum drain placed between muscle and periosteum. It is activated every 2–4 h, for 5 min only. A compression dressing is applied to the face for three days. I always applied an ice pack with compression for 24 hours, followed by warm fomentation dressings for 2–4 days and early mouth opening exercises. Every patient who undergoes masseter muscle reduction has to work hard to prevent scarring of the remaining muscle, by controlled mouth opening exercises for up to 3 months.

On the radiograph, the decortication result may look very uneven and irregular (Fig. 100j). Within five months the two sides look almost equal again (Fig. 100k). The main objectives are the patient's final symmetrical appearance and no remaining complications (Figs. 100l–n). To achieve absolute symmetry in the very asymmetric cases is not that easy. It is possible either to excise on one or other side, too much or not enough. It is not easy to explain this to the patient pre-operatively but it is wise to do so and have his/her understanding.

23.4

Principal Complications, How to Deal with Them and How to Avoid Them

23.4.1

Problems During Surgery

Damaging the Mental Nerve. It has already been discussed in the chapter on chin correction. The problems are the same as there.

Bleeding from the Masseteric Artery. It emerges through the semilunar notch and runs laterally to the muscle part to be resected. If this happens, one has to try to clip it, although it might be difficult. In this case tamponade for a longer period is contraindicated because of the necessity to achieve normal mouth opening again soon after surgery. It has never caused us a real problem. - I have never experienced any severe bleeding from behind the ramus.

The Masseteric Nerve Might be Cut. I have never encountered this or its consequences.

Damage to the Facial Nerve. It will probably not be diagnosed before the patient is awake again. It is unlikely that the main stem or all its branches are cut. I have never seen it. But complete facial palsy or a weakness has been reported when the patient wakes up. It can easily happen if the assistant pulls too hard on the soft tissues with his retractor. For that reason retractors with a downwardly bent end should not be used at all in masseter muscle reduction.

Damage to the Mandibular Nerve. It can occur when cutting off the lateral and inferior bony excess. Careful surgery should avoid this. Once it has happened not much else can be done other than approximating the ends and trying to do nerve-suturing.

Fracturing a Bur. It can easily happen when rotating bone cutting instruments are used. When the fractured piece is not found immediately, it is best to leave it and forget it, but of course the patient must be told.

23.4.2

Problems After Surgery

A Haematoma. It can develop in spite of the use of a compression bandage. It has to be evacuated. For that reason the patient must be seen the day after surgery.

Infection May Develop. However, I have never encountered it after this operation. Treatment would again be irrigation with antibiotic saline solution and systemic administration of antibiotics in a very severe case.

Trismus. It will occur in every case, though differing in degree, in spite of pre- and postoperative injections of a cortisone derivate and immediate postoperative ice dressings for the first 24 h, in addition to the compression bandage. If necessary, they are followed by 2–4 days of warm fomentation dressings. Warm fomentation dressings are a good aid in the early resorption of ecchymosis and postoperative oedema. Simultaneously, mouth opening exercises using stacked wooden spatulas must be done repeatedly during the daytime. An activator-like appliance for the night can help a lot not to lose the amount of mouth opening which was gained during the day. Normal mouth opening of at least 40 mm interincisal distance must be achieved and maintained for a few weeks.

How is a warm fomentation dressing applied? A warm moist napkin is placed onto the area of the operation, trauma or inflammation; it is then covered by a sheet of waterproof material, extending beyond its margins. The whole dressing is kept in place by a woolen shawl or a small towel. A small electric pad set at low heat is placed on top of all of this. This poultice is left in place day and night, with repeated moistening of the napkin. After 2–4 days the desired effect is achieved: oedema disappears, ecchymosis resorbs, and trismus is relieved as the musculature relaxes better. When there is circumscribed infection fomentation dressings usually accelerate resolution thereof or produce localized abscess formation. The effectiveness of this type of fomentation dressing is superior to any other type of poultice we have used, although it might be called “grandmother’s remedy”.

Facial Nerve Damage. It may only be diagnosed after the patient is fully awake. If it is caused through cutting a main branch of the nerve, the site of injury must be located and resutured. Electric stimulation can prove whether it is cut or there is just lack of nerve conduction due to overstretching. If the latter is the cause of facial nerve weakness or a complete lack of activity, function will return again spontaneously. However, it may take half a year or more. In any case the patient should be seen by a neurologist.

Insufficient Correction or a Bilaterally Unequal Result. It may be an indication to operate again, a few months after the first intervention. One must be careful not to excise too much muscle and bone.

Tongue Reduction

24.1

Historical Background

Orthodontists have long realized that the tongue can be an aetiological factor in abnormal tooth position. Many others, orthodontists as well as surgeons, were confronted with the tongue as a problem in relapse following their treatment. U. Rheinwald and R. Becker (1962) expressed the opinion that in every case of the then-called mandibular prognathism, reduction in the size of the tongue should be considered before the definitive surgery. In the late 1950s we did a series of presurgical tongue reductions before we repositioned the mandible by a bilateral sagittal splitting procedure. In the German literature, the first tongue reduction was reported by H. Pichler (1948), performed at the request of his orthodontist L. Petrik.

24.2

Definition and Classification of Macroglossia

I will define macroglossia as a tongue which is constantly or under certain conditions too large for that space of the oral cavity which is limited by the occluding rows of the teeth when they are normally positioned on the base of the jaw.

My classification of macroglossia is the following (M. Mommaerts 1987):

Idiopathic Macroglossia. This condition is rather rare. It causes protrusion mainly of the lower front teeth, producing interdental spaces, but can also shift the bicuspids and even the molars laterally, producing a circular crossbite. The tongue looks large, is rather voluminous, but only slightly rounded on the dorsum.

Macroglossia as a Sign of Acromegaly. Its reduction in size is as equally important before the correction of an acromegalic antemandibulism as is the removal of the causative pituitary gland adenoma. The tongue looks really voluminous.

Macroglossia in Cases of Down's Syndrome and Debility. In these cases the tongue has a rather typical form. It does not seem large or flat as in the genuine macroglos-

sia case. This tongue looks roundish with a rather high dorsum. Its consistency is harder than that in the two types just mentioned above. It can cause speaking problems and produce an open bite.

Functional Macroglossia. In the resting position nothing seems to be wrong with the tongue, but it presses with abnormal force against the teeth. The patient is often called a "tongue thruster".

Tumorous Macroglossia. The enlargement is due to a tumour or tumour-like condition within its musculature. Any kind of tumour in the tongue can produce such a macroglossia, a pure haemangioma or lymphoma and mostly a lympho-haemangioma, a neuroma, a fibroma or neurofibromatosis as in cases of von Recklinghausen's disease, etc.

Pseudo-macroglossia. This is that type of large, flat, flabby tongue which is often found within the large oral cavity in a case of macromandibulism or antemandibulism. It has no influence on the teeth or the position of the jaw. It has filled the space provided by the large jaw. The tongue does not need to be reduced in size when the mandible is pushed back and the oral cavity is reduced in size. It adapts to the new condition without any negative consequences.

24.3

Surgical Problems

As macroglossia was a well-known problem, several surgeons had reported their procedure to reduce the tongue size (Fig. 101a). Some removed just an oval wedge from the dorsum of the tongue. H. Pichler (1948) excised an additional oval piece from the tip. A rather large triangular piece of the tip of the tongue was excised by U. Rheinwald (1957). His technique shortened the tongue but did not reduce its breadth. H. Köle (1970a) used a rather narrow excision in the tongue's midline and additionally a triangular excision of the tip. I myself have excised an oval piece from the tongue's midline area and in addition a triangular piece in the front of the tongue.

We have published the procedure used, the results and the consequences (Egyedi and Obwegeser 1964).

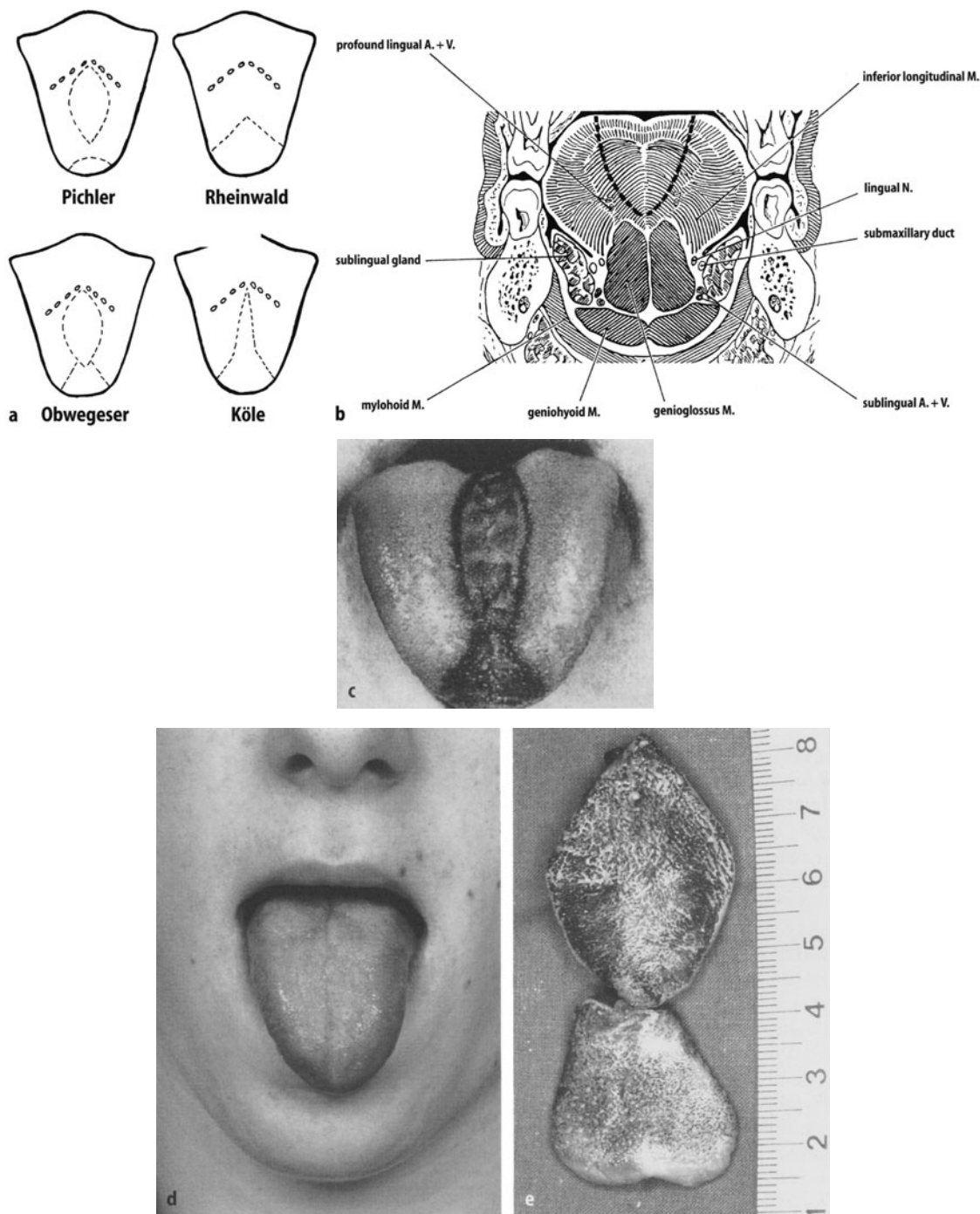


Fig. 101a–e. Tongue reduction. **a** Schematic illustration of the principal types of excision for tongue reduction. **b** Diagram of frontal section through the tongue with surrounding tissues in the molar region (from Corning 1909). **c** Large tongue with the desired amount of excision marked with blue. **d** The size and shape of the tongue 1 1/2 years after its reduction. **e** The size and shape of the excised parts of the tongue shown in Fig. 101c, d

Between 1957 and 1963 we performed tongue reduction on 21 cases, influenced by U. Rheinwald's (1957) philosophy on that subject. P. Egyedi has carried out the follow-up of our cases. He had also investigated cadaver tongues experimentally. We wanted to know why we had occasionally vascularization problems with the tips of the two halves of the reduced tongue. He found that this was most probably due to a sharp bend in the lingual artery towards our resection area.

When performing a tongue reduction, it is wise to remember its anatomy and in particular the location of the lingual artery in relation to the planned surgery (Fig. 101b).

After we had experienced partial necrosis of the tip of one or the other half in some cases, we reduced the breadth of that oval excision (Fig. 101c) and avoided cutting vertically down to the base of the tongue. The above mentioned sharp bend of the lingual artery, found on angiography of cadaver tongues, was the cause of it by being in the surgical field.

This procedure can produce a very nicely shaped tongue if it is not overdone. That means that not more, but rather less than a third of the tongue's width should be excised and the excision should be such that a good movable tip of the tongue will result (Fig. 101d). Care must be taken not to go too deep with the knife to the bottom of the tongue in the oval excision in the tongue midline. That means the knife should not go vertically down, but should be directed towards the midline of the tongue's lower surface in order to avoid cutting the lingual artery at its kink. It is astonishing the amount of tongue tissue that can be excised without interfering with its function (Fig. 101e).

24.4 Results

We were able to do a follow up on 20 out of 21 cases of tongue reduction, 7 of them had had a tongue reduction prophylactically. One of them ended up with a partial relapse of his presurgically existing open bite. In six cases, we did a tongue reduction when we thought that a relapse of the correction of an antemandibulism seemed to be developing. In one of them an unilateral relapse increased, in spite of the tongue reduction. The histology of the resected condyle proved the existence of condylar hyperactivity. The other cases remained satisfactory. In seven cases, the tongue size was reduced at the request of the orthodontic department. Two of them, one with an antemandibulism, the other with an open bite, remained as they were, in spite of additional orthodontic treatment.

There are two additional interesting cases which I want to mention. We did a mandibular set back operation on a 20-year-old female, using the sagittal splitting procedure. The patient had 6 weeks intermaxillary fixation and did not return for 6 months after removal of

the wire splints. The occlusion was the same as when she had left. When checking her, we saw that her large tongue was protruding through the bilateral gaps where the second bicuspid and first molars were missing. The crown and bridge department supplied her with a fixed bridge on each side. When she was called in for a check up after 9 months, it was found that on both sides the bridges were pushed laterally, but there was no change in the front tooth situation.

In the second case, a 28-year-old lady, a lower alveolar protrusion was present but there was no antemandibulism. The protruded alveolar segment was surgically repositioned into correct angulation with the anterior base of that mandible and with the upper teeth. Two plates were used to fix the repositioned alveolar segment in the new position. Within 6 months, the alveolus with its teeth was in its presurgical protruded position again. Now the patient's large tongue was reduced in size and then the lower anterior alveolus was again repositioned (see M. Mommaerts 1987).

24.5 As I Always Did It (P. Egyedi and H. Obwegeser 1964)

Normally, with the patient under naso-tracheal intubation anaesthesia, the tongue is gently pulled out, holding it with a towel clip inserted in its tip. The amount of oval excision in the midline, beginning just in front of the papillae and ending just behind the tip of the tongue, is marked with ink. Depending on the tongue's size 30–40% of the whole tongue width may be excised. In addition a V-shaped excision is marked, beginning on either side of the tip extending back to about the middle of the tongue (see Fig. 101c). Care must be taken to make a nice tip. For cutting the covering mucosa of the tongue I have given up using the electro-knife as the edges invert much more than when an ordinary knife is used. The bleeding is carefully arrested by electro-coagulation or ligation (preferably the latter). Then the covering mucosa is slightly undermined, as it otherwise inverts towards the tongue musculature. Resorbable material is used for adapting the muscle wounds. These stitches should not go too far inferiorly in order to avoid ligating the lingual artery. For closing the mucosa at the back of the tongue I prefer to use Supramid, placing some stitches of the mattress type. For approximation of the wound edges of the delicate mucosa on the inferior surface of the tip of the tongue this Supramid is also used, but of a very fine gauge. We leave the stitches in for 12 days.

24.6 Complications

During surgery the main problem was severe bleeding from the lingual artery. Although the vessel retracts into

the musculature it was clipped and ligated, resulting in partial necrosis of the tip of that half of the tongue.

After surgery we have experienced such massive swelling of the tongue that the patient could not close his teeth. Whenever we tried to evacuate blood through the suture line we had no success. So it must have been a type of parenchymal bleeding rather than a haematoma. This also happened in spite of careful control of haemorrhage during surgery. Within a few days the tongue always returned to the reduced size. We never experienced any infection. There was an occasional partial wound dehiscence. There were, of course, unpleasant results in those cases in which we had a partial tip necrosis of one or even both sides. The tongue looked asymmetrical and was rather hard and too short. There were speech problems for some days or weeks and the patients could never properly clean their vestibular sulci. After we became less radical with our oval excision, the final results did not have these unpleasant consequences.

24.7 Consequences

Based on my and my coworkers' experience I recommend that one should be rather conservative with tongue reduction. There are cases in which it should be recommended to the patient. The surgeon should keep in mind that a short, tethered tip of the tongue is a real disadvan-

tage for the patient, in speaking, in cleaning the teeth and the vestibular sulci with the tongue, and socially.

True, functional and tumorous macroglossia have more power to displace the teeth or even the alveolus than pseudomacroglossia has. It is my opinion that the tongue itself can rarely be the cause of a skeletal relapse after a setback operation for the correction of antemandibulism. When we reduced the tongue of every antemandibulism case prophylactically before the setback operation, we soon learned that this was totally unnecessary. Even in cases of true macroglossia the relapse may not be a skeletal relapse but an occlusal relapse because it is only the alveolus with the contained teeth which is tilted by the pressure of the tongue. Even screws and plates cannot prevent the relapse of a corrected ante-alveolism in cases of macroglossia, unless the tongue is primarily reduced in size.

Summarizing my experience with the tongue and its reduction, I have to say that there is no question that the tongue can have an influence on the position of the teeth and the alveolus. When functional or true or tumorous macroglossia is proven, then a tongue reduction should be advocated. Whenever one is in any doubt as to whether or not the tongue is or may be the cause of an occlusal or skeletal disturbance, I suggest performing the treatment first, be it orthodontic or surgical, and wait and see. If there is a tendency to relapse, the tongue should be reduced in its width and length by an experienced surgeon.

Instruments

The most clever surgical procedure may be difficult and time-consuming if the proper instruments are not available. As much as any operation technique will be altered and improved and modified through experience with it, the same is true for instruments which will undergo some changes and improvement, either by the originator of the procedure or by others. That's how progress is made.

For the surgeon, it is easy to improve his surgical technique. But he can hardly make the necessary instruments himself, alter them or develop new ones. He may have an idea of what kind of instrument he wants. But he does not always receive from the instrument maker what he thought he had ordered. The instrument maker has to understand how the surgeon wants to use the instrument. I have met only a very few really good ones who can grasp quickly the concept and details of what is requested and produce a usable product. Once the instrument is made according to the surgeon's requirements, he can evaluate whether or not that instrument is able to do what he expected. Very rarely is the very first the final one. That means that the instrument maker must be willing to work by trial and error.

The development of new instruments and their alteration is expensive for the instrument maker. Some manufacturers were not able to produce what I wanted and others very rarely produced it at the first attempt. When the person responsible for product merchandising in an instrument sales company feels confident to evaluate whether or not that instrument is going to sell, although having no conception regarding the use of the instrument and decides himself whether or not your requested instrument should be made, then every hour invested in the instrument you want is often wasted.

Twice, I took an idea to two different instrument-making companies and was told there was no call for that idea of mine. So it would not be developed. Later I found that the companies had produced my idea as their own, resulting in good sales for the company.

If you want an instrument to be reproduced in the quality you desire, you must have a written agreement with that company that you have the right to check your instrument's quality and design, if it is to be sold with your name attached to it. It is almost unbelievable what alterations instruments undergo when they are copied

but not checked by the surgeon who had developed them originally.

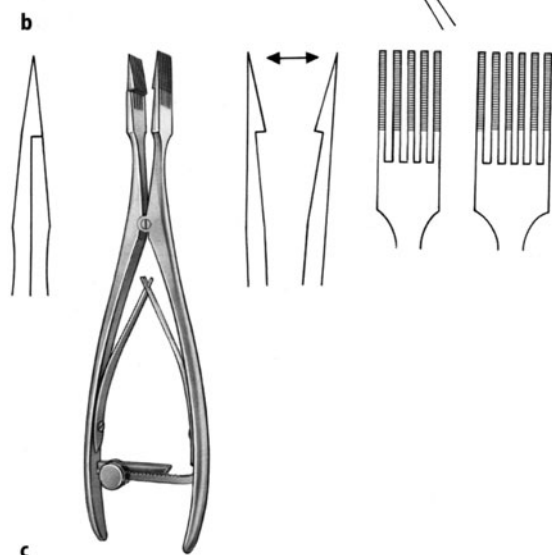
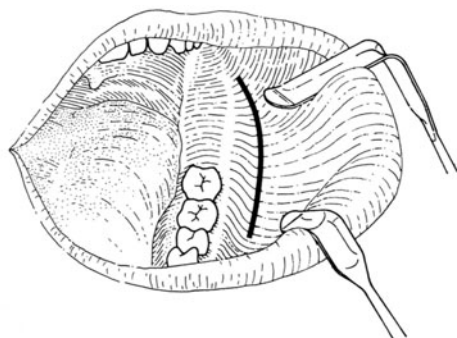
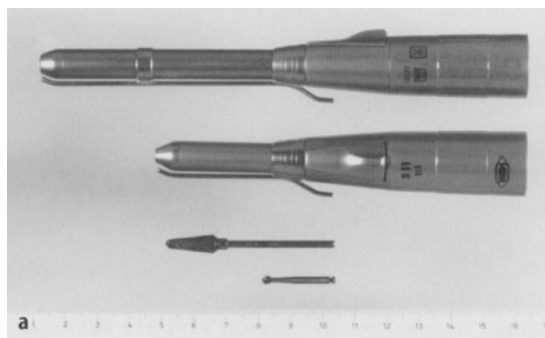
This chapter is not meant to become a catalogue of all the instruments which I had specially made for my type of surgery. I want to mention just a few of them which have made my surgery so much easier.

A Special Surgical Handpiece for the Electro-motor. It was needed in order to be able to use any rotating bur-like instrument available on the market with a shaft diameter of 2.35 mm. It had to be slim and so long, that while working in the depths of the mouth, the surgeon still has good visibility. The W & H Company in Austria was willing to develop it for me. It is a wonderful handpiece, which permits the use of burs with a very short as well as a normal or an extra long shaft. That means that any bur with an ordinary shaft diameter of 2.35 mm can still be used when its shaft is inserted only 10–12 mm into the handpiece. This is very advantageous for working in the depths. It also has the advantage that the holding system for the bur can be handled by the surgeon himself without his assistant's help. The W & H Company has supplied also the same type of bur-holding system to its already existing short surgical handpiece (Fig. 102a).

Indications for the use of the long surgical handpiece are any bone work in the depth such as removal of impacted wisdom teeth, osteotomies of the jaws, osteotomies of the orbits, any surgery on the cranio-facial skeleton, any reconstruction work as well as ENT precision bone work.

The same company has produced a very efficient and strong electro-motor for dental and other surgical purposes. It is called Elcomed[®] and can run from 200 up to 50,000 revolutions per minute. With the company's speed reduction handpiece, the speed can be reduced to approximately 0.2 turns per second, very useful for dental implant work. The high speed permits a special hard steel bur to be used like a fine paint brush. The cable and motor is fully autoclavable. It also has an automatic sterile water delivery system, which can be attached to a small tube on the surgical handpiece for simultaneous rinsing and cooling of the bone and the instrument while working.

Indications for the use of the electro-motor: Its power permits it to be used for working with any type of



saw, reciprocating as well as oscillating types, as long as the saw handpiece can be attached to an ordinary type of dental motor. The motor is so powerful that it can easily handle those power requirements. At my request, the company is arranging to produce handpieces for both oscillating and reciprocating saws. They should be available in the near future. I have also suggested the production of their own disposable saw blades for their new saw handpieces. They should be produced according to the requirements of the various specialties. In addition I have asked the company to produce a mini-reciprocating saw. .

Address of the Company: W & H Dentalwerk
Bürmoos GmbH, Austria, 5111 Bürmoos

The Gutter-shaped Soft Tissue Retractors. With an upwardly or downwardly bent end are of real advantage for work in the mouth, compared with plane retractors. The gutter-shaped form does not cause damage to the lips as do the ordinary retractors. Its upwardly bent end is ideal for holding the cheek away without damaging its mucosal covering while those with the downwardly bent end are used for retracting soft tissues as normally done. There are several types, differing in length and width (Fig. 102b).

The Bone Separator (Fig. 102c). It is an instrument which makes the sagittal splitting of the mandibular rami (see Fig. 87d) and also any required decortication so much easier. It is also used for the posterior and anterior downward fracture technique in the Le Fort I-type osteotomy (see Fig. 96e). In addition is an excellent instrument in cranio-facial surgery for mobilization of the middle third of the face after a Le Fort III-type osteotomy and also for mobilizing the orbital cones.

With the Maxillary Advancer (Fig. 102d and see Fig. 96g). With it the often difficult problem of mobilization of the maxilla is finally solved. With this instrument, the surgeon no longer needs to fear that a half or the whole maxilla is pulled completely out of the mouth when he has difficulty in mobilizing it or has to accept insufficient mobilization. It is also a very useful instrument for advancing the middle third of the face. Once all necessary osteotomy cuts are completed, with that instrument the surgeon can pull the piece of bone to be mobilized forward millimetre by millimetre.

Fig. 102a–d. Important special instruments. **a** W+H surgical handpieces which can hold any length of bur with a 2.35 mm diameter shaft, even the very short ones. The long handpiece is very useful for wisdom teeth removal and bone work in the depth of the operating field. **b** Gutter-shaped soft tissues retractors with upwardly curved ends. **c** Bone separator. It exists in two widths. It is very useful for several indications. It can be fixed in any opening position. **d** The maxillary advancer

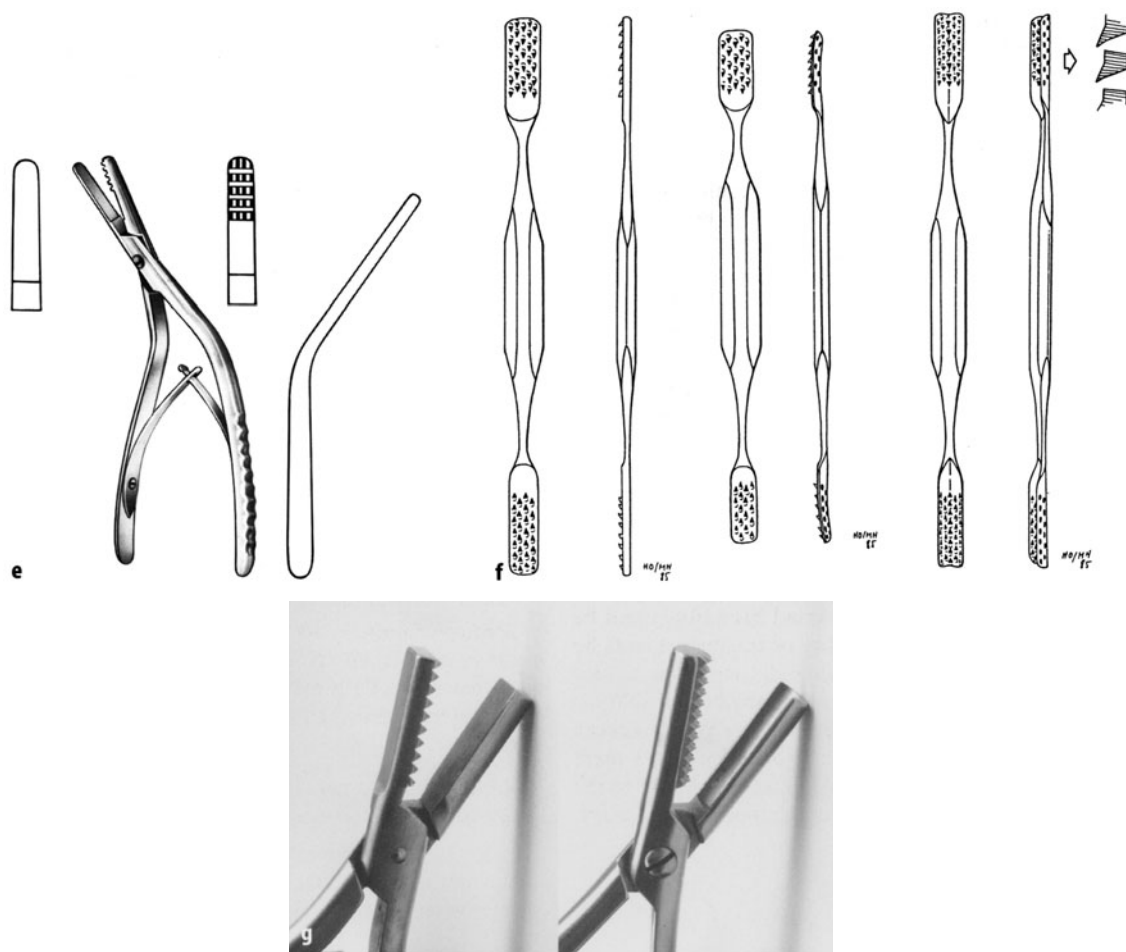


Fig. 102e–g. Important special instruments. **e** The nasal septum cutter. **f** The perforated nasal rasps. **g** The bone contouring forceps, round and flat

The Nasal Septum Cutter (Fig. 102e). With it it is intended to weaken the deviated septal cartilage without mutilating it badly by squeezing it as other instruments for the same purpose do. With it, many small cuts are produced in the area of the cartilage which eliminates the tendency to return to its original bent form. These little cuts will become fixed together by scars in their new position. It is, of course, necessary to have a tampon or soft rubber drains inserted in both nasal cavities to adapt the mucosa to the weakened septal cartilage to ensure its straight final shape.

The Perforated Nasal Rasps (Fig. 102f). They are not normal bone rasps which become clogged with bone mush after 2–3 pulls. They have sharp teeth to engage the bone surface and the mush is pushed through perforations which widen conically towards the back of the rasp. This prevents obstructing on the the

perforations and keeps the instrument working effectively.

The Bone Contouring Forceps. A flat and a round type exist. They are very useful for contouring a piece of soft bone to the shape the surgeon needs. Cortical bone cannot be bent. The instrument is ideal for contouring decorticated halves of ribs (Fig. 102g).

25.1

Where to Obtain the Instruments in Checked Quality

In any instrument catalogue for maxillofacial surgery there are instruments advertised as Obwegeser instruments. Some of them are worse than poor copies of my original instruments. Even in the well-known instrument-producing companies the shape and quality of the

instruments change within a few years. This is my experience. For that reason and for further improvement it is necessary that the surgeon checks, at intervals, all those newly produced instruments which are sold under his name. There are only two instrument-producing companies which want me to check their production of Obwegeser instruments. These two companies alone and their subsidiary companies hold the right to put my name on their products. They may distribute these instruments directly or sell them to other distributing companies. So, in addition to my name, there must always be the name of that manufacturing company on the instruments which I recommend.

In the USA and Canada my instruments were primarily distributed by the Walter Lorenz Company only. Walter Lorenz and I had a longstanding friendship which started in 1967. Literally overnight, he improved a few instruments for me so well that I could use them in a demonstration operation during a postgraduate course that was transmitted by TV into the lecture hall. I thanked him during the operation for his cooperation. All the attending surgeons wanted his address and he wanted me to check the quality of the instruments he was going to sell under my name. His name was a guarantee of quality. Before he passed away he had sold his company. With the new director of the Walter Lorenz Company the philosophy changed. When I was there last, in 1995, I was surprised what change some of the Obwegeser instruments had undergone. Quite a num-

ber of them were almost not usable for the purpose for which I had originally designed them. The new director was also not interested in my newer improved and the newly developed instruments. It is for that reason that the Walter Lorenz Company is not permitted now to put my name on any instrument they sell.

KLS in Jacksonville is now the only instrument distributing company in the USA which sells my instruments checked by me for their quality. The company's instruments are made in Tuttlingen, Germany.

Address: KLS-Martin L.P., P.O. Box 50249,
Jacksonville, FL 32250-0244, U.S.A.

Martin at Tuttlingen, Germany, a very large instrument distributing company, also distributes my instruments all over the world. It obtains the instruments from the same manufacturer as does KLS.

Address: Martin Medizin Technik, Ludwigsthaler
Strasse 132, Postfach 60, 78501 Tuttlingen, Germany

Medicon Company at Tuttlingen, is also producing my instruments, all of them. They want me to check what they sell as Original Obwegeser Instruments. That company has developed for me some of my newer instruments.

Address: Medicon eG, Postfach 4455,
78509 Tuttlingen, Germany

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